An investigation into the cause of insidious hypothermia occurring during immersion in lukewarm water, and of the mental consequences of hypothermia

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ABSTRACT

During the late 1970s, when oil exploration and extraction from the North Sea were at a peak, there was increasing concern about the number of episodes of unexplained confusion, loss of consciousness and deaths during dives.

Previous field measurements on divers had demonstrated that divers became hypothermic with little or no sensation of cold, despite suit heating using tepid water pumped from the surface.

This thesis describes laboratory experiments designed to document and to determine the cause of 'insidious' hypothermia. Initially, it was shown that uniform skin cooling in tepid water could produce subnormal body temperatures in all subjects tested, whether or not they had been acclimatised to cold.

This symptomless fall in deep body temperature could be reversed by further chilling the hands and feet using a separate water circulation system, while the rest of the body remained in tepid water. The rise in deep body temperature was shown to be due to an increase in metabolic rate caused by shivering, with cold-acclimatised subjects shivering less.

The main cause of 'insidious' hypothermia is therefore inadequate skin stimulation of thermoregulatory reflexes by lukewarm water, with previous cold water exposure further reducing responses.

The next series of experiments was designed to assess the impairment of memory and reasoning processes by cold, since most previous evidence had been inadequate or anecdotal. Psychological tests were administered during the unusual physiological circumstances on rewarming after cold immersion, where subjects felt warm and comfortable, but had a low or falling deep body temperature.

The results clearly showed that the ability to form new memories was seriously impaired even by mild falls in temperature, and that reasoning processes were greatly prolonged, although remaining accurate.

The current work has therefore successfully determined the cause of the hypothermia which occurs in lukewarm water, and has shown that mental abilities are seriously affected early in the development of hypothermia.

this thesis composed solely by myself and completed July 1987.

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INTRODUCTION

During the late 1970s, oil exploration and extraction from the North Sea were at a peak. Diving operations were an essential part of this. However, with the increase in diving activity, there seemed to be a disproportionate increase in hazardous incidents. Deaths averaged six yearly, mainly in the British sector. At least half of these were unexplained, and many other divers survived periods of unexplained confusion and loss of consciousness (Childs and Norman, 1978).

The major difference between North Sea operations and those elsewhere in the world was the sea temperature, seldom above 15°C even in summer at the surface, and below 10°C at depths of 50 m or more. On a field survey of divers' temperatures subsequent to dives breathing standard helium-oxygen, Keatinge, Hayward and McIver (1980) showed that a number of divers were returning with deep body temperatures around 35°C after dives of 1-4½ hours. Furthermore, they were often unaware of being particularly cold.

Diving suits were heated by employing the warm-water flooding system, where hot water was pumped from the surface to the suit via the diving bell, and exited over the hands and feet via the wrists and ankles. Water temperature was monitored in the bell, but not at the suit. Regulation of suit temperature depended on the report of the diver's feeling hot or cold. The field survey data had shown that it was not necessary for the suit heating to fail for divers to become hypothermic. There were similarities between the 'silent' or 'insidious' hypothermia experienced by these divers and a laboratory simulation, where a lean and fit young man had cooled progressively in lukewarm water at around 29°C without any serious sensation of cold (Hayward and Keatinge, 1979).

Further laboratory investigations were therefore planned. These had two main components. The first was to immerse further subjects in lukewarm water in order to determine the characteristics of progressive symptomless cooling and then manipulate the temperature over parts of the body surface to break up the uniform nature of the temperature stimulus. It was hoped that an adequate thermoregulatory response could thus be restored. Secondly, there were many anecdotal reports of confusion or altered consciousness with cold exposure, but no previous attempts had been made to define the deep body temperature at which these started to occur. Furthermore, there were only a few conflicting experimental studies of the nature of any mental impairment. Again, using the immersion laboratory to produce mild hypothermia in volunteers, this problem could be tackled by administering standard psychological tests with accurate measurement of responses under finely controlled conditions.

The work was undertaken by a small team under the direction of Professor W. R. Keatinge at The London Hospital Medical College and supported by a grant from the Science and Engineering Research Council.

The general experimental ideas were produced by Professor Keatinge, with detailing, execution, volunteer acquisition and data analysis performed by myself and aided by Miss S. R. K. Coleshaw, a postgraduate student who joined the project at the same time. Invaluable help with selection of psychological tests was given by Dr. H. M. Davis, clinical psychologist, of The London Hospital Medical College. Mr. P. J. Mincer, an undergraduate student from The North East London Polytechnic, provided a welcome pair of hands during immersion experiments. I am grateful to Mrs. I. Sampson for typing the manuscript and to Dr. J. K. Stothers for a critical reading of the contents.

REFERENCES

Childs CM, Norman JN (1978). Unexplained loss of consciousness in divers. Médicine Subaquatique et Hyperbare 17, 127-128.

Hayward MG, Keatinge WR (1979). Progressive symptomless hypothermia in water: posible cause of diving accidents. Br Med J 1, 1182.

Keatinge WR, Hayward MG, McIver NK (1980). Hypothermia during saturation diving in the North Sea. Br Med J 1, 291.

CHAPTER ONE

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INTRODUCTION TO EXPERIMENTS ON THERMOREGULATION IN THE COLD

Heat is lost from the human body in any cold environment because there is no such thing as a perfect thermal insulator. It is particularly important that there are physiological ways of conserving body heat during exposure to thermal stress such as cold water immersion, because the thermal conductivity of water is twenty times that of air.

Body heat is produced by all metabolising tissues, and is lost to the environment predominantly through the skin, and to a much lesser extent by expired air. In cold water, losses through the skin take place by conduction and convection (Burton and Edholm, 1955; Hardy and Dubois, 1938).

The main defence against such heat loss is thermal insulation. In a hairless animal like man, insulating clothing is of prime importance. The diver's dry suit traps a layer of poorly thermally conducting air around the body, or his wet suit traps a thin layer of water between the body and the poorly conducting neoprene foam of his suit. At greater depths, water pressure compresses both wet and dry suits so they are no longer good insulators, and either a hard suit is worn, or the soft suit is heated. If helium-oxygen is used, as in deep or prolonged dives, respiratory heat losses become considerable but this is another topic.

Physiological insulators are twofold. Subcutaneous fat is a poor conductor of heat, and in man is distributed mainly on the trunk, upper arms, and thighs. Heat loss from the forearms, lower limbs, hands and feet is controlled by altering the amount of warm blood circulating in the subcutaneous veins and skin arterioles. In the cold, in addition to a reduction in the temperature of blood reaching the hands and feet by countercurrent exchange of heat within the muscles of calf and forearm (Bazett et al, 1948; Burton and Bazett, 1936), there is an increase in vasomotor tone which produces constriction of subcutaneous veins and

skin arterioles (Bini et al, 1980a; Bini et al, 1980b; Edholm, Fox and MacPherson, 1957; Forster, Ferris and Day, 1946; Spealman, 1945; Zelig, 1969).

Burton formulated the concept of heat flow from central regions of the body through an insulating barrier (Burton, 1934). He produced a mathematical model where the body consists of a stirred "core" with a nearly uniform temperature, and a surrounding "shell" through which a temperature gradient exists between core and surface, with the human body being approximately 60% core and 40% shell. This thesis is not primarily concerned with modelling, but makes use of the concepts of core or "deep body" temperature, insulation, and conductance, the latter being a term derived for the amount of heat lost per unit area for a temperature gradient (see Chapter Two).

It is apparent that insulation can be varied according to the degree of peripheral vasoconstriction, but in adult man heat production in the cold can also be increased by shivering (Carlsen, 1954). The increase in metabolic rate caused by shivering can be quantified by measuring the increase in oxygen consumption (Spurr, Hutt and Horvath, 1957). Non-shivering thermogenesis occurs in some other animals, and has been well studied in rats (Dunhoffer, Saudy and Szegvari, 1964), but does not occur in adult man (Smith and Horowitz, 1960).*

The primary stimulus to the heat preservation reflexes of increased peripheral vasoconstriction and increased metabolic rate is cooling of the skin. Vasoconstriction occurs rapidly when the skin is cooled below 33°C, and heat loss from the limbs reaches very low levels once this has occurred (Cannon and Keatinge, 1960), although exercise in the cold increases this heat loss by both vasodilation in the exercising muscles and increased heat production by the muscles (Hayward and Keatinge,

1) * Thermogenesis here indicating brown fat metabolism rather than dietary thermogenesis.

1981). In contrast to the completeness of peripheral vasoconstriction once it has occurred, the amount of increase in metabolic rate in the cold depends on several factors. Carlson observed in 1954 that the time to onset of shivering in humans was related to the area of skin cooled: during exposure to cold air, covering the thorax when supine delayed the onset of shivering longer than covering the legs (Carlsen, 1954). Nadel (Nadel and Horvath, 1969; Nadel et al, 1970) exposed subjects to different air temperatures following a reduction of the deep body temperature by eating ice-cream. He demonstrated that the increase in metabolic rate thus produced was proportional to the coldness of the air below 33.8°C, and that the metabolic response was greater with lower core temperatures. Similarly, Nielson (1976) found that warming the skin of subjects, who had lost 1-2°C in deep body temperature while swimming in cold water, produced an immediate fall in oxygen consumption, even while deep body temperature continued to fall. Cannon and Keatinge (1960) investigated the factors governing the ability to maintain deep body temperature in cold water, and found that thin men stabilised their deep body temperatures at a lower level than fat men, and had a higher metabolic rate than fat men when stable. Both fat and thin men had increased metabolic rates when water temperature fell below 33°C, but those of the thin men were greater.

These findings showed that the response to cutaneous cooling depends on the area cooled as well as the degree of cooling, and that the metabolic response is reinforced by a fall in deep body temperature.

The ability to stabilise deep body temperature in cold water depends on the amount of insulation provided by peripheral vasoconstriction and subcutaneous fat, and the amount of heat generated by shivering above the normal metabolic heat production. Limb heat loss does not

differ much between fat and thin people, largely because limb vasoconstriction is very effective. To some extent, the lowest water temperature which a person can tolerate can be predicted from the mean subcutaneous fat thickness (see Chapter Two for details of measurement). However, it is not easy to predict the amount of shivering that may take place in an individual although there is a general relationship between the increase in oxygen consumption in the cold and the maximum oxygen uptake (VO2 max) when subjects are tested on a bicycle ergometer (Golden et al, 1979). Furthermore, the unpredictability lies not in the ability to generate heat, but whether or not the ability is used. For example, Hayward and Keatinge (1979) reported the case of a physically fit, thin, rowing cox whose deep body temperature fell below 35°C in water at 29°C, and yet in water at 26°C was able to increase his metabolic rate substantially.

There are two possibilities for a sub-optimal metabolic response to cold. Firstly, cold adaptation may alter reflex vasoconstrictor and metabolic responses, and secondly, if the water is lukewarm or tepid instead of undoubtedly cold, there may be inadequate stimulation of cutaneous cold receptors. These ideas are further explored below.

Acclimatisation to repeated local cooling of the hands tends to result in a raised threshold for cold induced pain, and the maintenance of warmer tissue temperatures by earlier local vasodilation. Cold vasodilation occurs in unacclimatised people at skin temperatures below 12°C (Lewis, 1930; Greenfield et al, 1951; Keatinge, 1957). This is due to direct vascular muscle paralysis by cold (Keatinge, 1958). Among those showing the acclimatised response to hand chilling are fish filleters (Nelms and Soper, 1962), army recruits training in cold climates (Keatinge and Evans, 1958), Gaspe fishermen of the Canadian arctic

(Le Blanc, Hildes and Heroux, 1960) and Eskimos (Miller and Irving, 1962). Another people, the diving women of Korea (Ama) have higher forearm muscle temperatures in winter, but reduced hand and finger temperatures. This is attributed to a more efficient venous return through muscle (Paik et al, 1972), and may be a consequence of whole body immersions causing intense skin and finger vasoconstriction, rather than the local reduction in vascular tone in the hands of fully clothed people. The latter is likely to be due to a reduced arousal, or habituation, to repeated hand chilling (Keatinge and Evans, 1958).

By way of contrast, evidence of an increased peripheral vasoconstriction as a long term adaptation to whole body cooling is given by Goldly (Goldly et al, 1938). He examined Australian Aboriginals, who were regularly cold exposed by sleeping in the cold desert air at night. Using the amplitude of radial artery pulsation as an indicator of peripheral vasoconstriction in both unacclimatised whites and aboriginals, he found that although both felt cold at similar skin temperatures, vasoconstriction occurred earlier in the aboriginals.

Skreslet and Aarefjord (Skreslet and Aarefjord, 1968) measured rectal temperatures and metabolic rates during acclimatisation by daily scuba diving in 2°C water. From these, they were able to deduce that after a month, there was a generalised increase in vasoconstriction of the extremities in response to a cold challenge. This increased response remained thereafter while repeated cold exposure took place. Their deduction was based on the findings that on the first cold exposure, there was both an increase in metabolic rate and rectal temperature, but that a month later the rectal temperature could be maintained by a much reduced rise in metabolic rate.

It seems, therefore, that part of the mechanism of cold acclimatisation is an increased tissue insulation caused by reduced blood flow to the extremities. However, in terms of whole body heat balance, changes in metabolic rate with acclimatisation may be more important. As mentioned above, Skreslet and Aarefjord found metabolic rate declined with repeated cold exposure. This in fact took place over the first two weeks. Primitive tribes such as the Australian Aboriginals, and Alacaluf Indians of Southern Chile (Elsner, 1963; Hammel et al, 1959; Hildes, 1963) allow their deep body temperatures to drift down during the night by 1-2°C and shiver little. Although it has been suggested that this lack of response to cold was due to malnutrition (Hammel, 1964), it is probably a genuine physiological response because it has been found in other subjects. For example, Skreslet and Aarefjord's divers were not malnourished. Hanna and Hong (1972) also describe a delay in the onset of shivering in regular scuba divers compared with non-divers exposed to cold water baths. Similarly, there was a delay in the onset of shivering in regular surfers compared with non-surfers when both were immersed in 19°C water (Rochelle and Horvath, 1978).

Changes in metabolic response and the degree of vasoconstriction of the extremities probably reflect the length of time over which acclimatisation has taken place. The initial response to cold immersion is one of hyperventilation and elevation of blood pressure (Keatinge and Nadel, 1965) but for immersions of 30 minutes or longer there is a sustained metabolic response and increased vasoconstriction (e.g. Cannon and Keatinge, 1960). With repeated immersions, or regular whole body chilling by cold exposure, a general pattern of response is discernable. The metabolic response to cold exposure persists but declines gradually over the course of weeks until by between six weeks and two months of cold

acclimatisation the metabolic rate does not remain elevated during cold. In parallel to this is a reduction of heat loss from the extremities with time. Similar patterns are seen in long-distance swimmers (Golden et al, 1979), and in arctic soldiers and explorers who frequently became cold and wet (Budd, 1965; Le Blanc, 1956; Livingstone, 1976). When reports of increased metabolic rate with exposure are examined (Elsner, 1963; Carlson et al, 1953; Hildes, 1963; Scholander et al, 1958), the feature of recurrent whole-body chilling is absent, and indeed the subjects often became fitter, i.e. their VO_2 max increased.

Similarly, the local early vasodilation of the hands seen in people acclimatised while fully clothed is a different response from that produced by whole body cooling, possibly because heat balance of the body is unaffected, and only small areas involved.

As to the question whether tepid water provides an inadequate cold stimulus to generate thermoregulatory reflexes whether or not a person is cold acclimatised, water at 29°C may well not provide such a stimulus. Thermoregulatory reflexes depend to a large extent on information from cutaneous themoreceptors, and various experiments indicate that the perception of temperature is not acute in tepid water. For example, thermal sensation during hand cooling in air is neutral in the range 29-32°C (Enander, 1982). In other words, at these temperatures the skin feels neither cold nor warm. Discriminating ability, measured by the ability to distinguish a difference in temperature between two thermodes on the forearm, is least between 27°C and 33°C (Erickson and Poulos, 1973), and between skin temperatures of 28°C and 37.5°C, there is a complete absence of any thermal sensation after 20 minutes (Kenshalo and Scott, 1966).

The puzzle to be unravelled hinges on Keatinge's observation that the rowing cox, who became hypothermic in water at 29°C, was still able to maintain his deep body temperature in colder water. Was his condition in the tepid water a result of repeated exposure to cold, or was he a victim of a thermoregulatory loophole in water at 29°C? Were his responses adequate for air exposure, but not for the high heat loss caused by water?

For divers using the warm water flooding system, the answers are important. If the flooding water is merely tepid rather than warm, are they going to cool and eventually become hypothermic, or can heat balance be maintained if the water is tepid as long as they avoid becoming acclimatised to cold?

REFERENCES

Bazett HC, Love L, Newton M, Sisenberg L, Day R, Forster R (1948). Temperature changes in blood flowing in arteries and veins in man. J Appl Physiol 1, 3-19.

Bini G, Hagbarth KG, Hynninen P, Wallin BG (1980a). Thermoregulatory and rhythm generating mechanisms governing the sudomotor and vasoconstrictor outflow in human cutaneous nerves. J Physiol 306, 537-552.

Bini G, Hagbarth KE, Hynninen P, Wallin BG (1980b). Regional similarities and differences in thermoregulatory vaso- and sudomotor tone. J Physiol 306, 553-565.

Budd GM (1965). Effects of cold exposure and exercise in a wet, cold, Antarctic climate. J Appl Physiol 20, 417-422.

Burton AC (1934). The application of the theory of heat flow to the study of energy metabolism. J Nutr 7, 481-497.

Burton AC, Bazett HC (1936). A study of the average temperature of the tissues, of the exchanges of heat and vasomotor responses in man by means of a bath calorimeter. Am J Physiol 117, 36-54.

Burton AC, Edholm OG (1955). Man in a cold environment. London, Edward Arnold.

Cannon P, Keatinge WR (1960). The metabolic rate and heat loss of fat and thin men in heat balance in cold and warm water. J Physiol 154, 329-344.

Carlsen LD (1954). The adequate stimulus for shivering. Proc Soc Exptl Biol 85, 303-305.

Carlsen LD, Burns HL, Holmes TH, Webb PP (1953). Adaptive changes during exposure to cold. J Appl Physiol 5, 672-676.

Dunhoffer S, Saudy F, Szegvari G (1964). Brown adipose tissue and thermoregulatory heat production in the rat. Nature 203, 766.

Edholm OG, Fox RH, MacPherson RK (1957). Vasomotor control of the cutaneous blood vessels in the human forearm. J Physiol 139, 455-465.

Elsner RN (1963). Comparison of Australian Aborigines, Alacaluf Indians and Andean Indians. Fed Proc 22, 840-842.

Enander A (1982). Perception of hand cooling during cold air exposure at three different temperatures. Ergonomics 25, 351-361.

Erickson RP, Poulos DA (1973). On the qualitative aspect of the temperature sense. Brain Research 61, 107-112.

Forster RE II, Ferris BG, Day R (1946). Heat exchange and blood flow in the hand. Am J Physiol 146, 600-

Golden FStC, Hampton IFG, Hervey GR, Knibbs AV (1979). Shivering intensity in humans during immersion in cold water. J Physiol 290, 48P.

Golden FStC, Hampton IFG, Smith D (1979). Cold tolerance in long distance swimmers. J Physiol 290, 48-49P.

Goldly F, Hicks CS, O'Connor WJ, Sinclair DA (1938). A comparison of the skin temperature and skin circulation of naked whites and Australian aboriginals exposed to similar environmental changes. Aust J Exp Biol 12, 29-37.

Greenfield ADM, Kernohan GA, Marshall RJ, Shepherd JT, Whelan RF (1951). Heat loss from toes and forefeet during immersion in cold water. J Appl Physiol 4, 37-45.

Hammel HT (1964). Terrestial animals in cold: recent studies of primitive man. Adaptation to the Environment 413 (DB Dill, Editor) (Washington: Amer. Physiol. Soc.).

Hammel HT, Elsner RN, Le Messurier DH, Anderson HT, Milan FA (1959). Thermal and metabolic responses of the Australian Aborigine exposed to moderate cold in summer. J Appl Physiol 14, 605-615.

Hanna JM, Hong SK (1972). Critical water temperature and effective insulation in Scuba Divers in Hawaii. J Appl Physiol 33, 770-773.

Hardy JD, Dubois EF (1938). Basal metabolism, radiation, convection and vaporisation at temperatures of 22-35°C. J Nutrition 15, 477-497.

Hayward MG, Keatinge WR (1979). Progressive symptomless hypothermia in water: possible cause of diving accidents. Brit Med J 1182-1183 (5th May).

Hayward MG, Keatinge WR (1981). Roles of subcutaneous fat and thermoregulatory reflexes in determining ability to stabilise body temperature in water. J Physiol 320, 229-251.

Hildes JA (1963). Comparison of Coastal Eskimos and Kalahari Bushmen. Fed Proc 22, 843-845.

Keatinge WR (1957). The effect of general chilling on the vasodilator response to cold. J Physiol 139, 497-507.

Keatinge WR (1958). The effect of low temperature on the responses of arteries to constrictor drugs. J Physiol 142, 395-405.

Keatinge WR, Evans M (1958). Modification of acute reflex responses to cold by brief training in cold climate. Lancet 2, 1038-1041.

Keatinge WR, Nadel JA (1965). Immediate respiratory reponses to sudden cooling of the skin. J Appl Physiol 20, 65-69.

Kenshalo DR, Scott HA (1966). Temporal course of thermal adaptation. Science 151, 1095-1096.

Le Blanc J (1956). Evidence and meaning of acclimatisation to cold in man. J Appl Physiol 9, 395-398.

Le Blanc J, Hildes JA, Heroux O (1960). Tolerance of Gaspe Fishermen to cold water. J Appl Physiol 15, 1031-1034.

Lewis T (1930). Observations upon the reactivity of the vessels of the human skin to cold. Heart 15, 177-208.

Livingstone SD (1976). Changes in cold induced vasodilation during Arctic exercises. J Appl Physiol 40, 455-457.

Miller LK, Irving L (1962). Local reactions to air cooling in an Eskimo population. J Appl Physiol 17, 449-455.

Nadel ER, Horvath SM (1969). Peripheral involvement in thermoregulatory response to an imposed heat debt in man. J Appl Physiol 27, 484-488.

Nadel ER, Horvath SM, Dawson CA, Tucker A (1970). Sensitivity to central and peripheral thermal stimulation in man. J Appl Physiol 29, 603-609.

Nelms JD, Soper DJG (1962). Cold vasodilation and cold acclimatisation in the hands of British fish filleters. J Appl Physiol 17, 444-448.

Nielson B (1976). Metabolic reactions to changes in core and skin temperature in man. Acta Physiol Scand 97, 129-138.

Paik KS, Kang BS, Han DS, Rennie DW, Hong SK (1972). Vascular responses of Korean Ama to hand immersion in cold water. J Appl Physiol 32, 446-450.

Rochelle RD, Horvath SM (1978). Thermoregulation in surfers and non-surfers immersed in cold water. Undersea Biomed Res 5, 377-390.

Scholander PF, Hammel HT, Anderson KL, Loyning Y (1958). Metabolic acclimatisation to cold in man. J Appl Physiol 12, 1-8.

Skreslet S, Aarefjord F (1968). Acclimatisation to cold in man induced by frequent scuba diving in cold water. J Appl Physiol 24, 177-181.

Smith R, Horowitz BA (1960). Brown fat and thermogenesis. Physiol Rev 49, 330-425.

Spealman CR (1945). Effect of ambient air temperature and of hand temperature on blood flow in hands. Am J Physiol 145, 218-222.

Spurr GB, Hutt BK, Horvath SM (1957). Shivering, oxygen consumption, and body temperatures in acute exposure of men to two different cold environments. J Appl Physiol 11, 58-64.

Zelig R, Mason DT (1969). Comparison of the reflex activity of skin and muscle veins in the human forearm. J Clin Invest 48, 1870-1877.

CHAPTER TWO

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COOLING IN 29°C WATER

COOLING IN 29°C WATER

The first part of the investigation into the problem of insidious cooling involved simple immersion in tepid (29°C) water. Was this an effect that could be produced in anyone?

For a complete characterisation of the thermoregulatory response to tepid water immersion, it was necessary to immerse an adequate number of subjects in water at a finely controlled temperature. Measurements of tissue conductance were made when local heat flows were stable, and the metabolic rate due to shivering calculated from oxygen consumption.

SUBJECTS

Six volunteers acted as subjects. All were indoor workers and non-divers with no recent unusual exposure to cold.

FAT THICKNESS

Before immersion, all subjects were medically examined, their heights and weights were noted, and fat thickness measured ultrasonically (Wells Krautkramer model USM 2F). Although a simple machine in concept, with a single peizoelectric probe for transmission and reception of tissue ultrasound, considerable practice was necessary for accurate and reproducible results, because the pressure exerted on the skin by the probe could induce large errors if not constant. Fat thickness was measured at 4 sites, corresponding to the sites used by Hayward and Keatinge (1981) to calculate mean subcutaneous fat thicknesses:

Front of middle of upper arm

Anterolateral point of middle third of trunk

20 mm to side of midline, middle of lower third of trunk

Anterior midline of middle of thigh

Mean fat thickness was calculated from the sum of these measurements in mm, as 1.308 + 0.181 x S for men (r = 0.97) and 1.933 + 0.168 x S in women (r = 0.99) (Hayward and Keatinge, 1981) (S = sum).

SURFACE AREA

This was calculated for each subject from height and weight, using the formula of DuBois and DuBois (1916):

$$\text{Surface Area } (m^2) = 0.007184 \times W^{0.425} \times H^{0.726}$$

where W is weight in Kg

and H is height in cm.

CALCULATION OF METABOLIC RATE

Expired air was collected at intervals for 3-6 minute periods, via a Douglas bag with mouthpiece and valves. Volume was measured by emptying the bag through a dry gas meter (Gallenkamp model GF 095) using a modified vacuum cleaner.

Oxygen content was measured by a paramagnetic analyser (Beckman model E2). This machine produced stable readings throughout the course of an experiment. Careful warm up and calibration with standard gases was necessary each morning, because calibration did not remain stable overnight.

The total volume was corrected for the amount of gas used in the O2 determination (0.5 L), and minute volume (Ve) calculated fron the cor-

rected volume of expired air and duration of collection. The minute volume was then corrected to standard temperature and pressure, where:

$$Ve\ (STP) = \frac{Ve \times \text{barometric pressure} \times 273}{760 \times \text{temp of air in pump}}\ \text{l/min.}$$

Oxygen consumption (VO_2) and metabolic rate were calculated using the Weir formula (Weir, 1949).

$$VO2 = Ve\ (STP) \times \frac{20.93 - \%O_2}{100}\ \text{l/min.}$$

$$\text{Metabolic rate} = (1.046 - 0.05 \times \%O_2) \times Ve\ (STP) \times 69.77\ \text{Watts}$$

Standardised metabolic rates were then used as Watts/m^2.

HEAT FLOW AND TISSUE CONDUCTANCE

Heat flow bands constructed in the laboratory (Gin, Hayward and Keatinge, 1980) from copper-constantan wire and neoprene sheeting were used. Each band was individually calibrated using a known temperature differential to correct for heat flow reduction due to the band itself (see appendix). Output from the bands was measured in μV using a digital voltmeter (Fenlow Electronics, type 105 input impedance. 500 m). Calculation of heat flow across the band was as follows:

$$H\ (\text{Heat flow in Watts/m2 from skin}) = \frac{1}{\frac{1}{KE} - \frac{1d}{dT}}$$

where E is the voltage output of the band in μV, dT is the difference between temperature of the body core and the environment in °C. K is a

constant for Watts/m^2.μV, and ld is band insulation (°C.m^2/Watt) calculated during calibration.

Bands were placed on the right side of the body around the hand, foot, forearm, calf, and posterior and lateral aspects of the middle third of the trunk, using thermally conducting grease (Eccotherm TC4) between skin and band, and waterproof tape, taking care only to cover the edges of the band with tape. Tissue conductance to each region of the body surface was calculated as the rate of heat loss measured locally by the heat flow band, divided by the difference between deep body temperature and external water temperature.

TEMPERATURE MEASUREMENT

A zero-gradient aural thermometer (Keatinge and Sloan, 1975 and Appendix), in which enough heating is supplied to the outer ear to eliminate local temperature gradients, was placed in the right external auditory meatus to measure deep body temperature. This device responds rapidly to temperature changes (less than a minute), unlike rectal thermometers, which may lag up to fifteen minutes behind changes in deep body temperature. However, care was required to ensure that the flexible tip did not impinge on the tympanic membrane, or was uncomfortable in other ways. If disturbed in use, the elimination of local gradients by pad heating took ten to fifteen minutes. Similarly, it was important to ensure that the thermometer was well warmed up and in situ at least fifteen minutes prior to immersion.

TANK TEMPERATURE

Tank temperature was thermostatically controlled to 0.1°C, but measurements of precise temperature were made frequently using a British

Standards Laboratory mercury thermometer. For the later experiments with boxes of water at different temperatures within the tank, this was particularly important. Box temperature could be easily calculated by placing an end to end insulated copper-constantan thermocouple, with a 99% response time of 1.5 s (manufactured in the laboratory) in the box, with the reference thermocouple in the tank, and μV output recorded on the digital voltmeter.

IMMERSION TANK

Immersion took place in a 4,000 l tank of stirred water. Water temperature was thermostatically controlled to 0.1°C by electrical heating and refridgeration units which were part of the tank installation. Immersions were planned in advance so that the tank temperature could be selected at least twelve hours beforehand and adequate time allowed for accurate heating and any necessary adjustment. Water was stirred by compressed air driven through three ducts. The subsequent water agitation directed streams of water on to the subject at approximately 1 m/s from different angles. Subjects were immersed to the neck, male subjects wearing only bathing trunks and female subjects a two-piece bathing suit, sitting on a slatted wooden seat. Each subject wore a safety rope attached to a hoist in case quick removal was needed; and was attached to the seat by a quick release knot. The seat attachment proved necessary to prevent floating and possible subsequent isometric exercise while trying to remain seated.

SAFETY

Apart from the hoist attachment, each subject was fitted with electrocardiograph leads on the right shoulder and fifth left intercost-

al space. Rhythm was displayed on a monitor throughout the experiment. Subjects were told they could leave the tank at any time if they became too uncomfortable.

IMMERSION PROCEDURE

Once the electrocardiograph leads, aural thermometer and heat flow bands were attached (this taking up to an hour), subjects climbed down a ladder into the tank, and remained seated for 180 minutes. Metabolic rate was measured prior to immersion and at least 4 times during immersion. Aural temperature was continuously displayed, and recorded at 5-minute intervals.

Heat flow readings were obtained by manually switching channels on the digital voltmeter, and were recorded at 10-minute intervals.

At the end of the immersion, subjects were helped out of the tank and rewarmed in the adjacent bath full of water at 41°C. Rewarming was complete in 20-30 minutes.

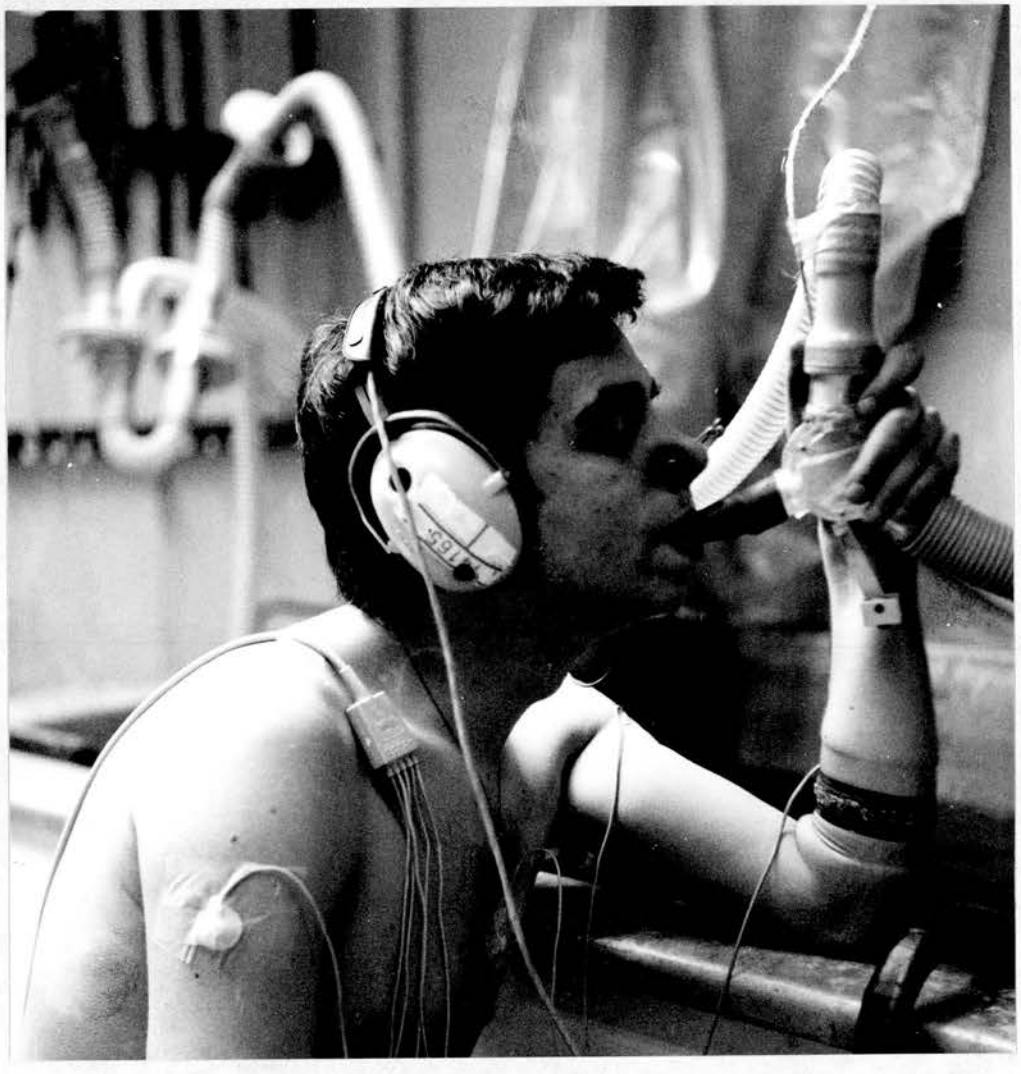
The experimental team comprised three staff, whose duties were (a) operation of the water pumps, if used (Chapter Four) and maintenance of tank temperature, (b) collection of expired air samples and measurement of oxygen content, (c) recording of temperature and heat flow data.

I was responsible for the running of the experiment and subject safety, and the other two team members were a PhD student at The London Hospital Medical College (SRKC) and an undergraduate student at the North East London Polytechnic (PJM).

RESULTS

Statistical comparisons between groups were made using the Student's t-test, and related t-tests for paired observations.

The Gallenkamp dry gas meter. In the foreground is a pump for hand and foot box water circulation.



Collection of expired gas prior to immersion. A heat flow band and electrocardiograph leads are well shown.

Fenlow Type 105 500 MΩ digital voltmeter.

TABLE 1: Subject anthropometric data.

SUBJECT	AGE	HEIGHT (m)	WEIGHT (kg)	SURFACE AREA (m^2)	MEAN SUBCUTANEOUS FAT THICKNESS (mm)
SC*	22	1.63	66.3	1.71	10.5
PM	22	1.86	67.5	1.89	6.4
NvS	29	1.83	77.0	1.99	7.5
RS	24	1.91	90.9	2.20	7.0
SP*	22	1.65	69.1	1.76	11.7
EG	28	1.68	65.5	1.74	9.3
Mean	25	1.76	72.7	1.88	8.7

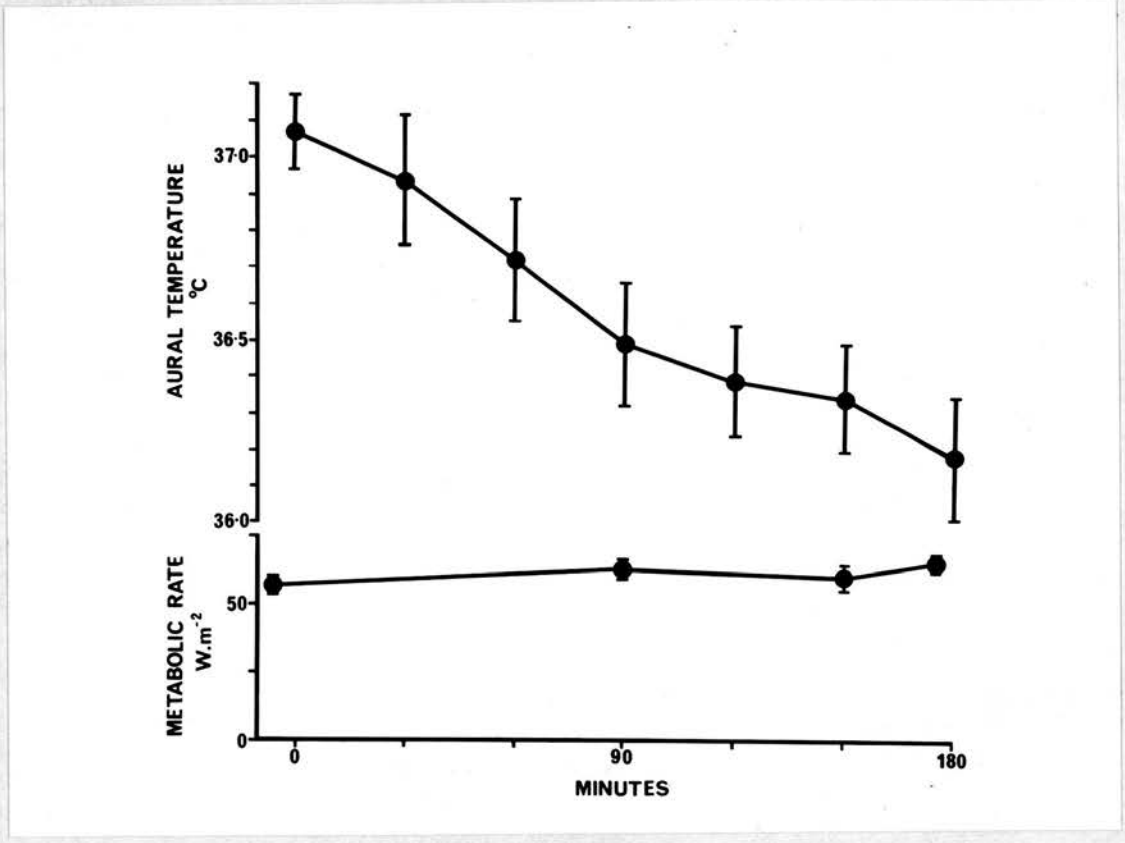


FIGURE 1: Deep body temperature and metabolic rate during immersion in water at 29°C. Values are means $\pm$ SE of experiments on 6 subjects, none of whom had recently been exposed to cold.

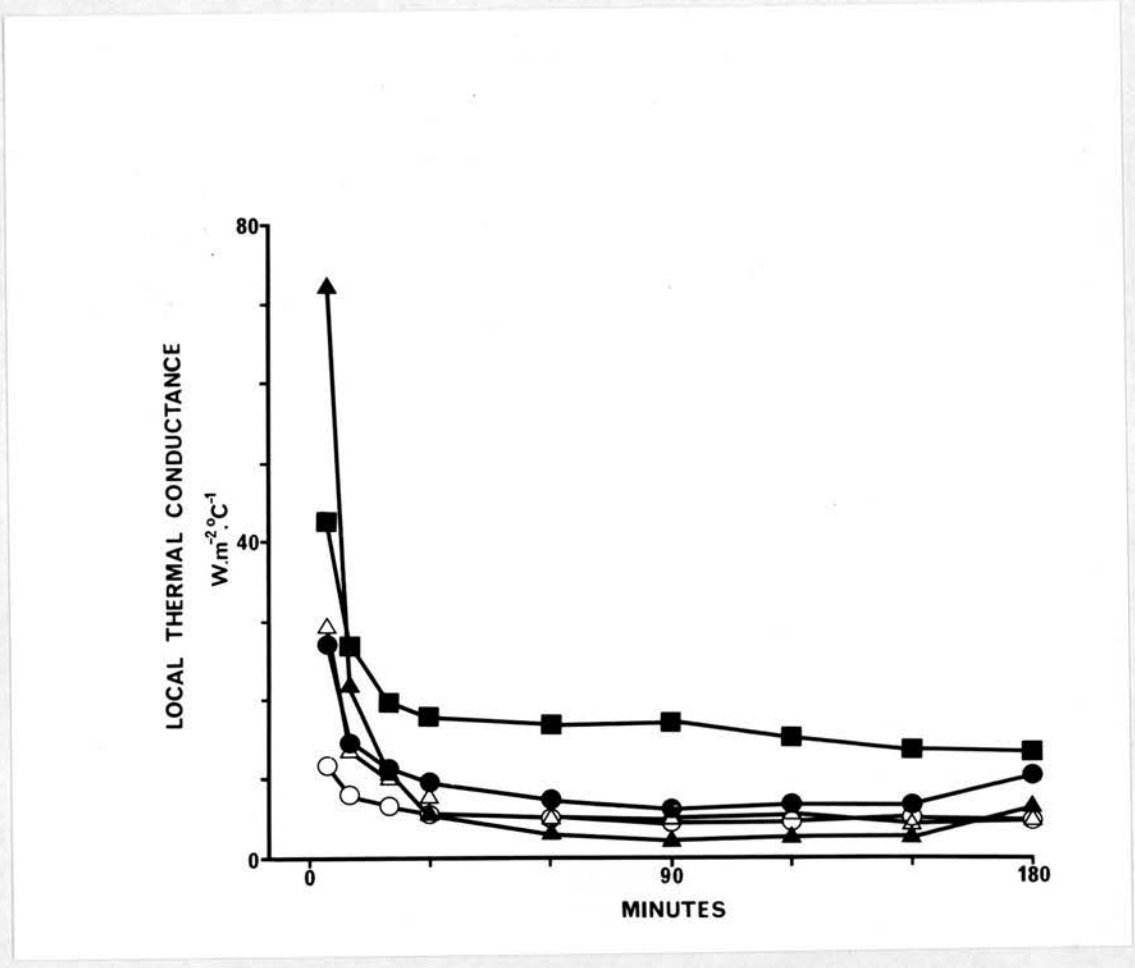


FIGURE 2: Tissue conductance during immersion in water at 29°C. Values are means ± SE for same 6 subjects as in Figure 1. ■, trunk; ▲, hand; ●, forearm; △, foot; o, calf. SEs at 180 min ($W.m^{-2}.°C^{-1}$); trunk, ± 4.3; hand, ± 1.5; forearm, ± 1.7; foot, ± 0.9; calf, ± 0.3.

Figure 1 shows that their deep body temperature fell throughout the immersion, but metabolic rate rose little. The average fall of temperature over the 3 hour period was 0.9 $\pm$ 0.17°C, the lowest body temperature reached in any subject was 35.6°C. Falls in temperature were generally larger in the thinner subjects; correlation coefficients between fall in temperature and inverse of the individual's mean subcutaneous fat thickness were 0.88 for the four male subjects and 0.71 for the four male and two female subjects together but did not reach statistical significance at the 0.05 level with these subjects alone. Metabolic rate with the subjects sitting quietly before immersion was 50.7 $\pm$ 3.3 W /m^2; it rose only slightly to 60.6 $\pm$ 3.4 W/m^2 90 min after the start of the immersion, and to 65.4 $\pm$ 2.8 W/m^2 just before the end of the immersion ($P < 0.05$). The subjects shivered only slightly and intermittently during the immersion and reported little sensation of cold.

Figure 2 shows that during the first 30-60 minutes of immersion regional heat losses were at high levels, which were largely attributable to loss of stored heat from peripheral tissues. Heat losses then became fairly stable at levels and indicated tissue conductances that were substantially lower in the limbs than in the trunk.

DISCUSSION

These results confirm that it is possible to induce falls in deep body temperature during immersion in water at 29°C. This happened in all six subjects. The range of temperature fall being 0.2°C to 0.8°C after 60 minutes, and 0.5°C to 1.4°C after 180 minutes. Similar results were obtained for twelve subjects during the next part of the experimental series (Chapter Three) with a range of 0.1°C to 0.7°C after 60 minutes.

The rate of fall of temperature in these subjects was largely related to the subcutaneous fat thickness, with a correlation coefficient between fat thickness and fall in temperature of -0.71 ($p > 0.05$). The lowest temperature reached after 180 minutes of immersion was 35.6°C, in a subject with a mean subcutaneous fat thickness of 6.4 mm.

The rowing cox studied by Hayward and Keatinge (1979) was leaner, with a mean subcutaneous fat thickness of 3.6 mm. After 112 minutes of immersion in water at 29°C, he was found to have a deep temperature of 34.7°C.

The key point concerning the immersion of the rowing cox was not that he cooled to such an extent, because all of the subjects continued to cool over the duration of the immersion, but that he possessed the capacity to prevent his deep temperature falling so low, and yet did not utilise it until he was immersed in colder water. During immersion in water at 29°C, his metabolic rate was 115 Watts/m^2 during the last 10 minutes, and he progressively cooled. However, subsequent immersions in progressively colder water showed that his metabolic rate rose to 220 Watts/m^2 in 22.8°C water, at which temperature he was able to stabilize his deep body temperature at 35.8°C. At water temperatures below this, he was not able to stabilise his deep body temperature, because the metabolic rate rose little more than in 22.8°C water, and insulation was unchanged.

Similarly, subjects in the experiments in 29°C water failed to increase their metabolic rate. Whether they were capable of doing so is the subject of the next chapter, but briefly, further local cooling of the limbs was found to provoke both an increase in metabolic rate and cessation of the previously steady fall in deep body temperature in 29°C water.

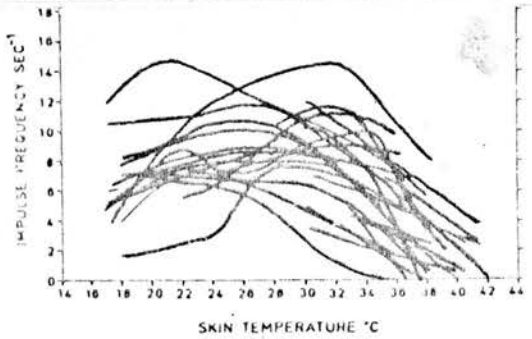
In other words, a skin temperature of 29°C does not provide an adequate stimulus to thermoregulatory reflexes to prevent deep body cooling in water.

The main reason for the development of this "insidious hypothermia" is failure to shiver. Subjects felt reasonably comfortable and were seen to shiver little. Metabolic rate rose little. On the other hand, there was probably an adequate reduction in skin blood flow to the limbs, because heat loss from these regions was very low. It has been shown that there is an increase in sympathetic nerve vasoconstrictor activity in the human forearm when the skin temperature falls below 33°C (Bini et al, 1980a and b), which thus appears to be the temperature threshold for initiation of skin vasoconstriction.

It is likely that the characteristics of cutaneous cold receptors are responsible for the inadequate shivering response to lukewarm water. Electrophysiological recordings of nerves supplying temperature sensitive skin have defined distinct responses to a change in temperature, the "dynamic" response, and responses to steady skin temperatures, the "static" response (Duclaux, Schafer and Hensel, 1980; Dykes, 1975; Hensel, 1973; Hensel, 1974; Iggo, 1969). These appear to apply in both animals and humans.

In contrast to the dynamic response of cutaneous cold receptors, which provide precise information on the size, rate, and direction of temperature change, the properties of cold receptors to steady temperatures are much less sensitive. Although skin temperatures of 25-29°C have generally been found to produce maximal rates of summated discharge from groups of cold receptors, the activity of individual cold receptors at various steady temperatures have poorly defined peaks spreading over several degrees. Thus individual receptors are poor

transducers of static temperatures. Information on static temperatures is probably furnished by the patterns of discharge which include receptors whose maximal firing rate is outwith the range provided by the majority of receptors. In the case of uniform skin temperatures of 29°C, although the majority of receptors will be activated, the information they carry is likely to be less than at more extreme temperatures (see diagram below).



Static discharge frequencies of 32 single cold fibers from the infraorbital nerve as a function of constant temperatures of the nose.

'Cold receptor activity at various temperatures' after Hensel (1973)

Normally, the face and hands are exposed, and the feet colder than limbs or trunk. Exposure of the hands and feet may usually be an important factor in generating overall thermoregulatory responses, because of their exposure to environmental temperature. Normally little heat is lost from these regions in the cold because of efficient counter-current heat exchange in the limbs (Bazett et al, 1948; Spealman, 1945), thus providing an ideal skin surface for temperature appreciation. Although the face is also usually exposed, it is less suitable as a site capable of initiating responses to cold because it is a region of high blood flow and heat loss (Froese and Burton, 1957). If the skin temperature is uniform, as in immersion experiments, the normal skin temperature differential is removed. This is an unusual circumstance, and one to which the thermoregulatory system may not be adapted. This is more fully discussed in Chapter Three.

A further possibility for impaired thermal sensation during water immersion is a direct effect of water on the receptors. These are probably superficially situated (Hensel, 1974) and one may expect osmotic or ionic changes in the skin to alter responses. PJ Mincer investigated these possibilities in this laboratory. His subjects were seated in a chair with both forearms immersed in water baths. The experimental arm was in direct contact with the water, while the control forearm was at the same temperature, but separated from the water by a single layer of "clingfilm". Difference in thermal sensation was measured by adjusting the temperature of the control water bath until both arms felt equally warm or cold. He found that soaking for three hours, either in freshwater or seawater, or at temperatures of 15°C, 22°C, 29°C and 38°C, did not alter thermal sensation significantly (van Someren et al, 1982). In other words, the cutaneous thermoreceptors appear to be immune to osmotic or ionic changes in the skin.

The next chapter describes experiments where the progressive deep body cooling in 29°C water is interrupted by altering the uniform skin temperature with localised cooling.

REFERENCES

Bazett HL, Love L, Newton M, Eisenberg L, Day R, Foster R (1948). Temperature changes in blood flowing an arteries and veins in man. J Appl Physiol 1, 3-19.

Bini G, Hagbarth KE, Hynninen P, Wallin BG (1980a). Regional similarities and differences in thermoregulatory vaso- and sudomotor tone. J Physiol 306, 553-565.

Bini G, Hagbarth KE, Hynninen P, Wallin BG (1980b). Thermoregulatory and rhythm-generating mechanisns governing the sudomotor and vasoconstrictor outflow in human cutaneous nerves. J Physiol 306, 537-552.

Dubois D, Dubois EF (1916). Clinical calorimetry: a formula to estimate the approximate surface area if height and weight be known. Arch Intern Med 17, 863-871.

Duclaux R, Schafer K, Hensel H (1980). Responses of cold receptors to low skin temperatures in the nose of the cat. J Neurophysiol 43, 1571-1577.

Dykes RW (1975). Cooling of steady and transient temperatures by cutaneous "cold" fibres sensing the hand of monkeys. Brain Research 98, 485-500.

Froese G, Burton AC (1957). Heat losses from the human head. J Appl Physiol 10, 235-241.

Gin AR, Hayward MG, Keatinge WR (1980). Method for measuring regional heat losses in man. J Appl Physiol 49, 533-535.

Hayward MG, Keatinge WR (1979). Progressive symptomless hypothermia in water: possible cause of diving accidents. Brit Med J 1182-1183, 5th May.

Hayward MG, Keatinge WR (1981). Roles of subcutaneous fat and thermoregulatory reflexes in determining ability to stabilise body temperature in water. J Physiol 320, 229-251.

Hensel H (1973). Neural processes in thermoregulation. Physiol Rev 53, 948-1017.

Hensel H (1974). Thermoreceptors. Ann Rev Physiol 36, 233-249.

Iggo A (1969). Cutaneous thermoreceptors in primates and subprimates. J Physiol 200, 403-430.

Keatinge WR, Sloan REG (1975). Deep body temperature from aural canal with servo-controlled heating to outer ear. J Appl Physiol 38, 919-921.

Spealman CR (1945). Effect of ambient air temperature and of hand temperature on blood flow in hands. Am J Physiol 145, 218-222.

van Someren RNM, Coleshaw SRK, Mincer PJ, Keatinge WR (1982). Restoration of thermoregulatory response to body cooling by cooling hands and feet. J Appl Physiol 53, 1228-1233.

Weir JB de V (1949). New methods for calculating metabolic rate with special reference to protein metabolism. J Physiol 109, 1-9.

CHAPTER THREE

THE EFFECT OF HAND AND FOOT COOLING

Having demonstrated that it is possible to insidiously induce hypothermia in lukewarm water, the next step involved an investigation into a way of stimulating the return of a thermoregulatory response.

If this "passive" hypothermia were due to a lack of peripheral stimulation, then if the skin were stimulated by cold, a reversal of central cooling may be predicted. Further information could be obtained by measuring the responses of a group of cold-inexperienced people and comparing these with a group of people regularly exposed to cold.

SUBJECTS

Twelve people volunteered as subjects. Six were indoor workers with no recent cold exposure, and six were amateur subaqua divers who had dived for approximately 1.5 hours a week wearing unheated suits (both wet- and dry-suits) in water at 4-15°C during the three months before the experiments.

Heights and weights were measured, surface area calculated using the du Bois formula (Dubois and Dubois, 1916), and mean subcutaneous fat thickness calculated. The latter was calculated in a similar manner to that in Chapter Two, using the measurements obtained ultrasonically at four sites over the body.

The divers included one woman and the non-divers two women. The divers were aged 21-43 years (mean $\pm$ 30, SE $\pm$ 3.6 year), compared with 19-28 years (24 mean $\pm$ SE $\pm$ 1.4) for the non-divers. They were a little taller (1.76 $\pm$ 0.04 m) than the non-divers (1.71 $\pm$ 0.04 m), and heavier (72.7 $\pm$ 3.0 kg and 64.4 $\pm$ 4.8 kg), with a greater mean subcutaneous fat thickness (8.2 $\pm$ 1.09 mm compared with 6.7 $\pm$ 0.62 mm). None of the differences between the two groups was statistically significant.

IMMERSION PROCEDURE

The apparatus and general laboratory conditions were identical to those used in the previous experiments described in Chapter Two. However, immersions were modified so that the hands and feet could be held at different temperatures from the rest of the body. The main interest lay in keeping the hands and feet colder than the trunk and limbs, because this is the normal pattern of skin temperature when clothed.

This was achieved by constructing a pair of perspex boxes, one of which enclosed the hands, and the other enclosing the feet (Fig. 1). Circular holes enabled the hands and feet to be placed in the boxes, and soft latex seals at the wrists and ankles prevented movement of water between the boxes and the tank. Care was taken in assessing the seal size for each subject, and the hole size was altered until there was no sensation of pressure by the subject, and no skin blanching. This was necessary to exclude any effects which may have been caused by inadvertently restricting the skin blood flow. After preliminary trials, the boxes were reduced to as small a size as possible while allowing a stable separate water temperature to be maintained within. (Volumes of 22.5l for the foot box and 14.5l for the hand box). It was found that the time required to change the water temperature of larger boxes took anything up to ten minutes, which seemed unacceptably long. Using the smaller sized boxes reduced the time to between 3-5 minutes. Shorter times were not considered necessary because measurement of heat flows demanded steady-state conditions. The time taken to achieve stable regional heat flows varied with different subjects, and took between 30 and 60 minutes. Box water temperature was measured with a copper constantan thermocouple (99% response time 1.5 sec) suspended in the

TABLE 1: Subject anthropometric data for hand and feet cooling experiments.

SUBJECT	AGE	HEIGHT (m)	WEIGHT (kg)	SURFACE AREA (m^2)	MEAN SUBCUTANEOUS FAT THICKNESS (mm)
SH	26	1.76	86.0	2.11	8.2
PB	22	1.69	60.0	1.69	4.5
TS	22	1.63	56.0	1.59	5.3
MR	19	1.78	69.8	1.87	6.8
SL*	24	1.65	57.0	1.62	7.4
VvS*	28	1.64	57.5	1.62	8.1
Mean (non-divers)	24	1.71	64.4	1.75	6.7
RG	21	1.85	69.8	1.92	5.3
GP	26	1.72	68.2	1.80	4.8
TB	36	1.74	76.4	1.91	8.5
AR	43	1.86	85.0	2.10	8.4
SS	28	1.73	73.0	1.86	11.0
PS*	26	1.63	63.6	1.68	11.0
Mean (divers)	30	1.76	72.7	1.88	8.2

* = female

centre of the box, with the reference thermocouple in the tank, and output measured in μV on the Fenlow high resistance digital voltmeter.

Hand and foot movement were not restricted beyond the seals, and free circulation of water round the whole of the skin surface within the boxes ensured by allowing the fingertips to rest only on a transverse bar, and the soles of the feet on a slatted surface.

A hydraulic reservoir and circuit distinct from the main tank apparatus was used to supply the hand and foot boxes (see Fig. 2). Basically, a household bath was filled with water to act as a reservoir, and small electric pumps circulated water round both boxes in series and back to the reservoir. Hand and foot box temperature was monitored with thermocouples and reservoir temperature adjusted to maintain this. While this was a simple task if 29°C water was circulating, it took the full attention of one team member to accurately monitor temperatures and stir ice water into the reservoir accordingly when colder temperatures were used in the boxes. Care was taken to avoid fluctuation in reservoir and box temperature during steady state measurements but the box temperature could only be kept between 1°C each side of 12°C. The main tank thermostat automatically compensated the tendency of the tank to cool while cold water was circulating in the boxes, and fluctuations in tank temperature were never more than 0.1°C.

Aural temperature was recorded at five minute intervals using the zero-gradient thermometer. Hand, foot, forearm, calf and trunk heat flows were measured using heat flow bands, and expired air was collected prior to, during and after hand and foot cooling.

After 70-90 minutes, when heat loss from the hands and feet had stabilised, so that valid estimations of regional conductance could be made, cold water at 12°C $\pm$ 1°C was circulated through hand and foot

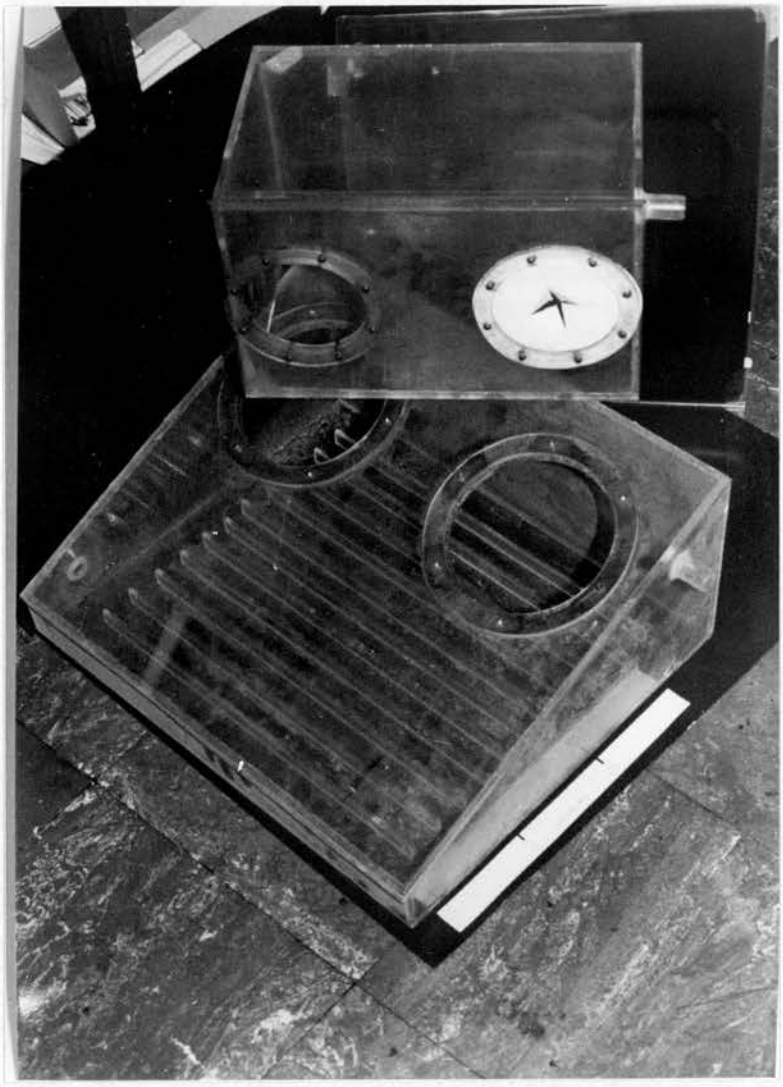


FIGURE 1

Hand and foot cooling boxes.

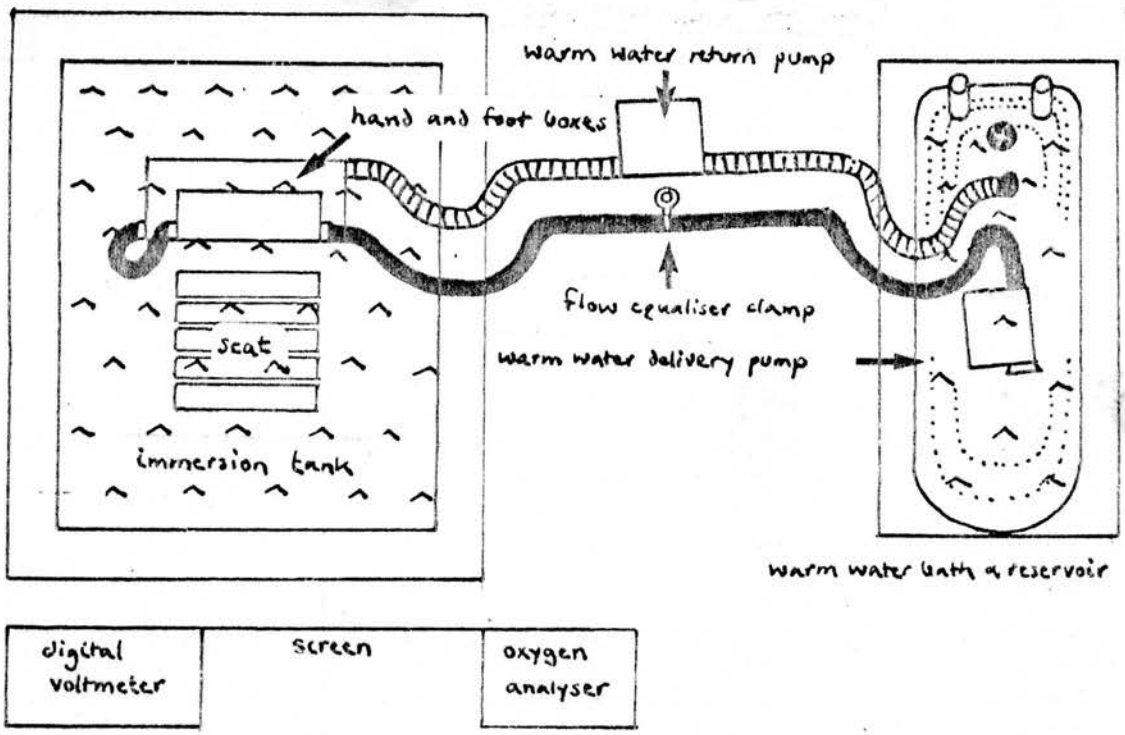


FIGURE 2

Hand and foot box circulation.

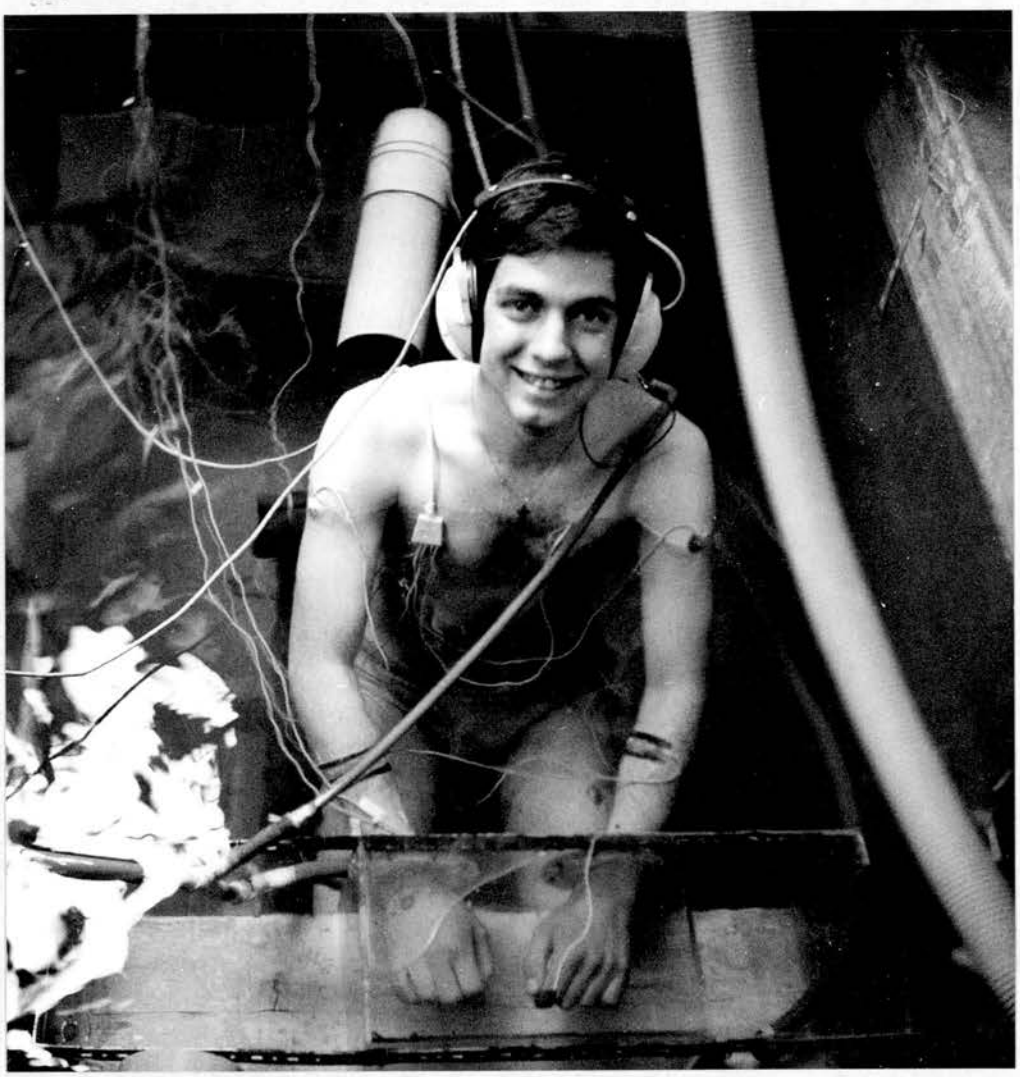


FIGURE 3 (a)

Subject instrumented and immersed during hand and foot cooling experiment.

boxes. After heat loss from the hands and feet had again stabilised (30-60 minutes), they were returned to water at 29°C ± 0.1°C for a further 30 minutes.

At the end of this time subjects were helped out of the main tank and rapidly rewarmed in the hot bath at 40-42 C.

RESULTS

DEEP BODY TEMPERATURE WHILE COOLING IN 29°C WATER

Figure 3 (b) shows that, in water at 29°C, the deep body temperatures of the divers and non-divers fell at similar mean rates of approximately 0.4°C/Hr. Rates of fall were faster in thinner than fatter subjects among the non-divers; correlation coefficients between rate of fall and the inverse of mean subcutaneous fat thickness were 0.78 for the male non-divers and 0.66 for all of the non-divers during the first 70 min of immersion at 29°C. When results for the male non-divers and the male controls of the previous experiments (also non-divers) were combined for the first 70 min of immersion at 29°C (total 8 subjects), the relationship between fall of deep body temperature and inverse of mean subcutaneous fat thickness was significant ($P < 0.05$, $r = 0.70$). No significant relationship was found between fall of deep body temperature and subcutaneous fat thickness in divers; for the male divers $r = 0.33$, and for all of the divers $r = 0.03$.

When the hands and feet were cooled in water at 12°C after 70-90 min of immersion at 29°C, deep body temperature of the non-divers ceased to fall and the rose slightly but significantly ($P < 0.05$) from 36.6 ± 0.2 to 36.8 ± 0.2°C by the end of 30 min; that of the divers ceased to fall but did not rise significantly. When the hands and feet

were returned to water at 29°C, the falls in deep body temperature were resumed, at a rate of approximately 0.6°C/Hr, in both divers and non-divers.

METABOLIC RATE

Before immersion, with the subjects sitting quietly, metabolic rates were similar in the divers and non-divers, 41.0 $\pm$ 2.5 and 42.9 $\pm$ 3.8 W/m^2 respectively (Fig. 3 bottom). After 70-90 min of immersion in water at 29°C metabolic rates had risen significantly in both groups ($P < 0.05$) but by only 26% in the divers compared with 45% in the nondivers; there was little obvious shivering and little sensation of cold in either group. Metabolic rates were then significantly lower in the divers than the non-divers. Five minutes after the start of hand and foot cooling, the metabolic rate of the divers had risen to 54% above resting level and that of non-divers to as much as 125% above resting level. Both of these increases were significant ($P < 0.05$), and the metabolic rate of the divers remained significantly lower than that of the non-divers. The non-divers all shivered markedly during cooling of the hands and feet, while the divers shivered noticeably less. All subjects, but particularly the non-divers, complained of both local cold sensation in the hands and feet and a general feeling of cold at this time. Metabolic rate, shivering, and sensations of cold then declined in both groups while the hands and feet remained at 12°C, but in non-divers they remained significantly above the previous level throughout the time that the hands and feet were cooled.

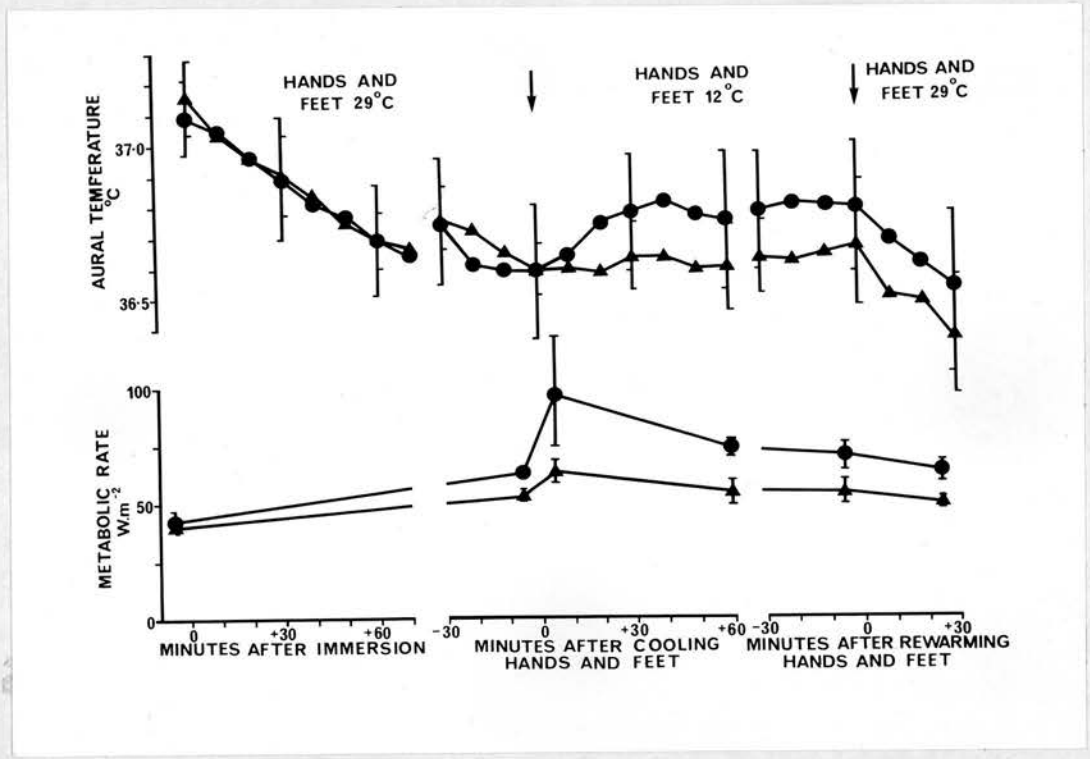


FIGURE 3 (b) Effect of cooling hands and feet in 12°C water, during general immersion in water at 29°C, on body temperature and metabolic rate. ● means ± SE, 6 unacclimatized subjects; ▲ means ± SE, 6 divers in cold-water training.

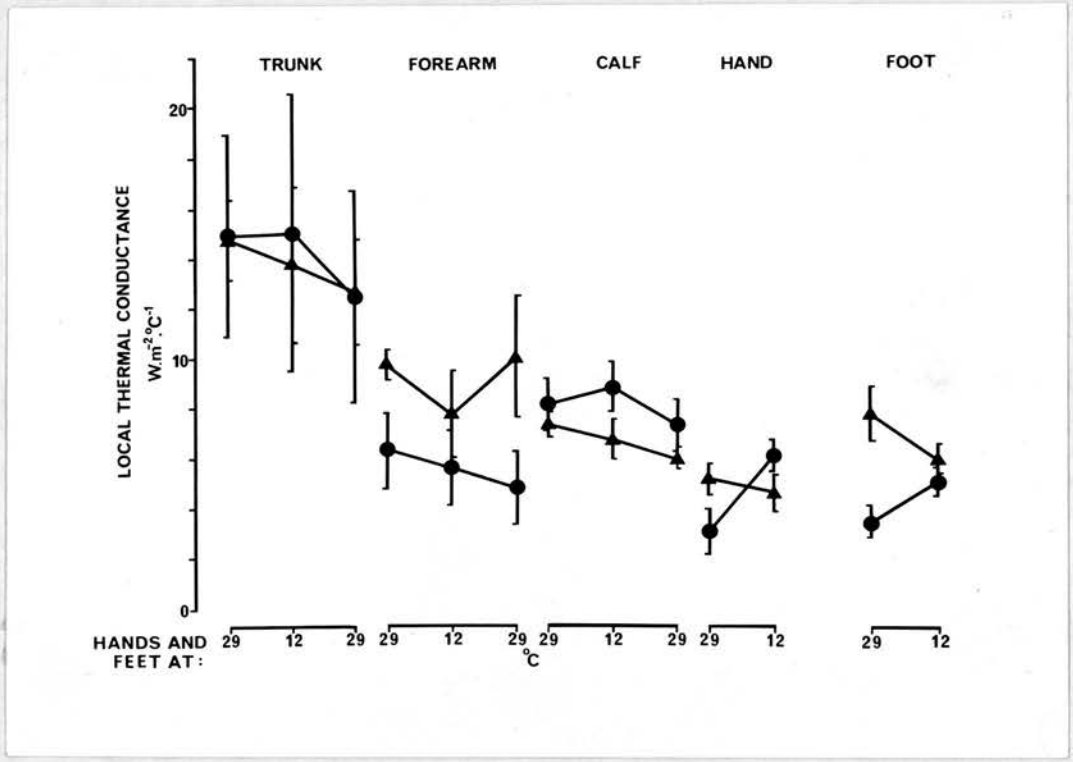


FIGURE 4: Effect of cooling hands and feet in 12°C water, during general immersion in water at 29°, on tissue conductance. Symbols as in Figure 3; same experiments on same subjects as in Figure 3. Values are stable values reached at end of periods with hands and feet in water at 29°C, then 12°C, then 29°C. Stable values were not reached for hands and feet in short second exposure to 29°C.

TISSUE CONDUCTANCE

Tissue conductances did not alter greatly on cooling the hands and feet in water at 12°C or on returning them to water at 29°C (Fig. 4). In general, tissue conductances tended to decline during cooling of the hands and feet in divers and to rise in the non-divers. The changes were small and generally not significant, but in divers conductance to the forearm was significantly less ($P < 0.05$) during cooling of the hands and feet than the mean conductance there before and after the cooling.

DISCUSSION

During the development of "insidious" hypothermia, the fall in deep body temperature can be halted or reversed by further cooling the hands and feet. The following discussion centres round the mechanisms of this phenomenen, and the patterns of cold sensation necessary to produce it. The ways in which this response is altered by previous cold exposure are then discussed.

Further cooling of the hands and feet in unacclimatised subjects undergoing 29°C immersions caused an increase in metabolic rate between one and a half times and double the metabolic rate seen while uniformly cooling in 29°C water, with only minor increases in heat loss. Thus there is no doubt that an increase in heat production is the major reason that deep body temperatures rise or cease to fall. Shivering is the cause for such an increase in heat production, and subjects were seen to shiver more during hand and foot cooling.

For the experimental conditions studied here, it appears that there is a limit to the amount by which metabolic rate can be increased.

Similar increases were seen if the hands and feet were cooled at 12°C or at 20°C (see Appendix). Cooling of the hands and feet at 25°C, however, increased the metabolic rate by much less in the same subjects.

From the present data, it is not possible to tell whether the important factor causing this increase is the hand and foot skin temperature alone, or whether the difference between peripheral limb temperature and trunk and proximal limb temperature is more important. However, this distinction may be artificial because the range of water temperatures causing insidious hypothermia is probably only 1-2°C either side of 29°C. This latter deduction arises from a consideration of cold thermoreceptor properties outlined in chapter one, where cold sensation is minimal in this particular temperature range.

For whole-body cold immersions it is known that the rise in metabolic rate is proportional not only to the water temperature, but also to the fall in deep body temperature (Cannon and Keatinge, 1960). Whether the drive from cutaneous cold receptors has a maximum which is constant below a "threshold" temperature is unknown, because there is usually also a reduced deep body temperature in cold immersion experiments. Certainly, the perceived intensity of cold stimulation of the skin is independent of internal body temperature (Mower, 1976), but the "hedonic rating", ie. the preferred most comfortable skin temperature, is markedly dependant on deep body temperature (Mower, 1976; Cabenac, Massonnet and Belaiche, 1972), with cold water being found more unpleasant if deep body temperature is also low.

A further influence on the perceived intensity of a cold stimulus is the temperature of the skin prior to the stimulus, with colder preceding skin temperatures inducing a more intense sensation of cold than the same stimulus applied to warmer skin (Ebaugh and Thauer, 1950). The

latter is likely to be a manifestation of neuronal or receptor adaptation to the ambient temperature. It therefore seems likely that the sensation of cold is proportional to the reflex drive to shiver, but this at present is an assumption.

The area of the hands and feet covers about 12% of the body surface (Dubois and Dubois, 1916). The hand and foot cooling experiments were designed to stimulate the reflex drive to shiver, and not to determine the minimum effective area capable of causing this. It is known that the sensation of cold depends on the total area stimulated, and is directly related to intensity (Kenshalo, 1976; Rozsa and Kenshalo, 1977). It is quite probable that there is a relationship between area cooled and increase in metabolic rate.

A further factor may be the site of cold stimulation. Peripheral "cold spots", i.e. sites where a sensation of cold can be elicited, are less dense on the hands and feet than the skin on either the face or trunk (Dollenbach, 1927). It may be more difficult to induce an increase in metabolic rate by hand and foot cooling than trunk cooling, and this relatively weaker drive may be abolished completely by lukewarm temperatures, with subsequent 'insidious' hypothermia.

The physiological properties of cutaneous thermoreceptors have been well described in the literature but the structure is not well known. The end organ of the receptor is not visible by light microscopy. Electron micrographs of the single receptive fields on the face of the cat show a thin myelinated axom dividing into several unmyelinated terminals within the stratum papillare. The receptive endings which protrude a few microns deep into the basal epidermal cells contain numerous mitochondria as well as an "axoplasmic matrix" with five filaments and microvesicles (Hensel, 1973; Hensel, Andres and v. During, 1974). The

specific human equivalent is likely to exist, but it has also been proposed that the smooth muscle of skin arterioles is also part of the temperature-sensing system (Kenshalo, 1976). Smooth muscle contraction to direct cold being the transducing mechanism, and subsequent deformation of innervating neurones communicating the stimulus. If this were the case, one would not expect much of a metabolic response to face and trunk cooling since these areas remain relatively vasodilated, and are areas of high heat loss in the cold (Hayward and Keatinge, 1981). It would be easy to design such an experiment, but for practical purposes the value of such information would be limited. Heat loss would be increased by face and trunk cooling, unless only a tiny area, perhaps only a few inches square, was effective. Even so, we know that the insidious hypothermia occurring in lukewarm water can be reversed by the simpler expedient of mildly cooling the hands and feet.

In this series of experiments, the temperature of the water in the hand and foot boxes could be changed in three minutes or less. The hands and feet remained in the cooling boxes for approximately an hour. The main stimulus to the skin of these areas would have been constant, with an initial stimulus due to the changing water temperature. The receptor response to a change in temperature contrasts with that to a constant temperature described in Chapter Two, where a large number of receptors are sub maximally stimulated. For cold receptors, there is a "dynamic" increase in firing when the temperature is being lowered, and a transient decrease when it is being raised.

This dynamic cold receptor response is related to the strength of the stimulus, and adapts in about thirty seconds. The threshold for a response to temperature change is small, about 0.1°C. The maximum dynamic responses are found in the temperature range 25-29°C (Bini

et al, 1980; Hensel and Boman, 1960; Iggo, 1969; Kenshalo and Duclaux, 1977; Molinari and Kenshalo, 1977).

One would expect, therefore, an increase and then a decline in the metabolic response to cooling.

In these experiments this was so. The initial rise in metabolic rate with hand and foot cooling declined after an initial rise, but the metabolic rate remained elevated above pre- cooling levels when a uniform tepid skin temperature was re-established.

There are two further points about receptor stimulation by peripheral cooling. If the hands and feet are cooled, leaving the wrists and ankles at 29°C, zones of thermal contrast are created in these areas, ranging from 17°C at the coldest temperatures to 4°C at the mildest used (see Appendix). There is evidence that the response of peripheral neurones is different at thermal gradients. Kenshalo and Gallegos (1967) describe the frequencies recorded from a specific cold sensitive fibre when cooling local areas of a monkey leg. Having established a number of local "cold spots", probably subserving a single afferent fibre, they found that the summed frequencies of fractions of the total number of cold spots stimulated were greater than when all the cold spots were simultaneously stimulated. Their explanation was that temperature differences between receptors on different dendrites of the same afferent neurone produced summation of frequencies by addition of generator potentials. If this were so, one would expect thermal contrasts to be preferentially accentuated. The maintenance of peripheral stimulation may therefore not be a function of area, but rather be related to the number of "cold spots" which are divided by the demarcation line between skin at different temperatures.

This hypothesis could easily be tested by attaching a cold "bracelet" of piped water to the wrists or ankles during tepid water immersion. This would have the added practical benefit that the fingers need not be chilled.

Secondly, although the sensation of cold is primarily dependent on peripheral receptors, there is evidence that the raw information is processed at a central level before the sensation is appreciated. For example, appreciation of a change in skin temperature is more prolonged than the dynamic response of a primary cold fibre (Kenshalo, 1976). Adaptation of the discharge of a primary cold fibre is complete in about 30 seconds, while adaptation of sensation to a similar fall takes tens of minutes. Further evidence is the demonstration by Rozsa and Kenshalo (1977) that the ability to discriminate small degrees of cooling of the human forearm is improved if the other forearm receives a simultaneous identical stimulus. It is not known if this is a cord or a cortical function.

Cortical evoked responses in the human to a skin cooling pulse on the thumb confirm cortical representation of peripheral cold sensitivity, with similar properties to the dynamic cold receptors of the monkey, followed by longer latency and ipsilateral responses (Chatt and Kenshalo, 1979). Rapid transit of basic thermal information from periphery to cortex followed by information of a modified kind would presumably maximise the cortical ability to discriminate temporal and spatial stimuli. There may be other influences on the afferent information, such as habituation to cold. It is important that altered responses to cold exposure are accounted for by a central mechanism, because the peripheral receptors probably do not undergo longterm adaptation. Recordings from the cold receptors of cold acclimatized cats show no

alteration in response, even after as much as five years acclimatization (Hensel and Schafer, 1979).

The divers, who were used to cold exposure, showed some differences from unacclimatized subjects in their response to cooling of hands and feet. Although the mean deep body temperatures of both groups ceased to fall, the method of achieving this was not identical in both. Divers decreased their heat loss from the limbs further, and increased their metabolic heat production little. The others increased heat production markedly, and lost more heat than previously. This state of cold adaptation in the divers is agreement with the final state of cold adaptation proposed by Aaresfjord and Skreslet (Chapter One).

Habituation to cold, ie. a lesser arousal to the same stimulus, is probably a major part of the explanation for the decreased metabolic response, because although all the subjects felt cold and shivered, the divers said they usually deliberately suppressed shivering as much as they could during a dive to reduce interference with the dive and prolong gas supplies.

Reduced O_2 consumption with repeated brief cold exposures has been observed in volunteer naval ratings immersed in 15°C water for eight successive days for 30 minutes (Keatinge and Evans, 1961). A more complex pattern of a daily increase in metabolic rate on exposure with a subsequent reduction below the metabolic rate achieved on the previous exposure was seen in lightly clad men in 6°C air for 7½ hours a day for days (Keatinge, 1961).

During the latter experiments the metabolic rate was found to increase before cold exposure, and this was probably due to apprehension. Reduced apprehension by habituation is the probable explanation for the higher threshold for cold vasodilation with cold acclimatization

(Chapter One). However, this cannot be the explanation for the tendency to reduce limb heat loss with repeated whole body cold exposure found by Aarefjord and Skreslet, and seen, although not to a significant degree, in our experiments. The explanation for reduced limb heat loss with repeated exposure may be that seen in the Ama (Chapter One), where the limb countercurrent heat exchange becomes increasingly efficient. Whether this is due to vascular capacitance changes, or microvascular shunting, or to a net reduction in heat loss by a decreased amount of shivering is not known. The explanation does not have much of a practical importance, however, because limb thermal conductances become extremely small on cold immersion both in unacclimatized and acclimatized subjects (Hayward and Keatinge, 1981).

In summary, the insidious fall in deep body temperature induced by lukewarm immersion can be reversed by supplying additional cold stimulation to the skin. Extra heat is generated by shivering, and heat losses from the cooled hands and feet remain small. The degree of extra cold stimulation required is small, and the spatial pattern may be as important as the area. With cold acclimatization there is less shivering, but sufficient occurs to prevent the deep body temperature falling further.

REFERENCES

Bini G, Hagbarth KE, Hynninen P, Wallin BG (1980). Thermoregulatory and rhythm-generating mechanisms governing the sudomotor and vasoconstrictor outflow in human cutaneous nerves. J Physiol 306, 537-552.

Cabenac M, Massonnet B, Belaiche R (1972). Preferred skin temperature as a function of internal and mean skin temperature. J Appl Physiol 33, 699-703.

Cannon P, Keatinge WR (1960). The metabolic rate and heat loss of fat and thin men in heat balance in cold and warm water. J Physiol 154, 329-344.

Chatt AB, Kenshalo DR (1979). The afferent fibre population mediating the thermal evoked response to skin cooling in man. Exptl Neurol 64, 146-154.

Dollenbach K (1927). The temperature spots and end organs. Am J Psychol 39, 402-427.

DuBois D, DuBois EF (1916). Clinical calorimetry: a formula to estimate the approximate surface area if height and weight be known. Arch Intern Med 17, 863-871.

Ebaugh FG, Thauer R (1950). Influences of various environmental temperatures on the cold and warmth thresholds. J Appl Physiol 3, 173-182.

Hayward MG, Keatinge WR (1981). Roles of subcutaneous fat and thermoregulatory reflexes in determining ability to stabilise body temperature in water. J Physiol 320, 229-251.

Hensel H (1973). Cutaneous thermoreceptors. Handbook of sensory physiology Vol II, 79-110. Heidelberg, New York: Springer.

Hensel H, Andres KH, v During M (1974). Structure and function of cold receptors. Pflugers Arch 352, 1-10.

Hensel H, Boman KKA (1960). Afferent impulses in cutaneous sensory nerves in human subjects. J Neurophysiol 23, 564-578.

Hensel H, Schafer K (1979). Activity of cold receptore in cats after longterm adaptation to various temperatures (Abstract). Pflugers Arch 379, R56.

Iggo A (1969). Cutaneous thermoreceptors in primates and sub-primates. J Physiol 200, 403-430.

Keatinge WR (1961). The effect of repeated daily exposure to cold and of improved physical fitness on the metabolic and vascular response to cold air. J Physiol 157, 209-220.

Keatinge WR, Evans M (1961). The respiratory and cardiovascular response to immersion in cold and warm water. Quart J Exptl Physiol 46, 83-94.

Kenshalo DR (1976). Correlations of temperature sensitivity in man and monkey: a first approximation. In: Sensory functions of the skin in primates. Ed.Y.Zotterman. pp.305-330 Pergamon.

Kenshalo DR, Duclaux R (1977). Response characteristics of cutaneous cold receptors in the monkey. J Neurophysiol 40, 319-332.

Kenshalo DR, Gallegos ES (1967). Multiple temperature-sensitive spots innervated by single nerve fibres. Science 158, 1064-1065.

Molinari HH, Kenshalo DR (1977). Effect of cooling rate on the dynamic response of cat cold units. J Expt Neurol 55, 546-555.

Mower GD (1976). Perceived intensity of peripheral thermal stimuli is independent of internal body temperature. J Comp Physiol & Psychol 90, 1152-1155.

Rozsa AJ, Kenshalo DR (1977). Bilateral spatial summation of cooling of symmetrical sites. Perception and Psychophysics 21, 455-462.

CHAPTER FOUR

INTRODUCTION TO EXPERIMENTS ON MEMORY AND COGNITIVE FUNCTION DURING HYPOTHERMIA

Confusion and unconsciousness are known to occur with profound hypothermia. Accounts of shipwrecks and behaviour of the chilled survivors consistently relate irrational behaviour or collapse.

James Currie, a Liverpool physician, recorded a sailor's account of a December shipwreck at the mouth of the River Mersey in 1797 (Currie, 1797).

> "The master of the ship, Capt. Scott, a native of North-Carolina, and about forty years of age, died first. As they were in the dark Mr. Amyat could not see his countenance; but he was at first alarmed by hearing him talk incoherently, like one in the delirium of fever. By degrees, his voice dwindled into a mutter, and his hearing seemed to fail. At length he raised himself up in a sort of convulsion, in which he continued a few seconds, and then fell back dead on the deck."

Currie described the captain as "strong and healthy, in the flower of life, early inured to cold and hardship, and very vigorous in both body and mind" and makes clear that his death was a result of cold rather than injury. Over a century later, a similar account was written about the wreck of the "Foyledale" in Valparaiso during a storm on June 2nd 1903 (Lubbock, 1932).

> "It was now about 5 am, and the captain's wife who, for the past hour, had been trying to cheer up her husband and encourage the men in the rigging above her, began to grow exhausted, and her brave voice gradually grew fainter until it could no longer be heard. The captain, who had been taking the weight of every sea on his broad back, was in a like state, dazed and choked by salt water and almost beaten senseless."

This captain, however, survived, and regained consciousness several hours after rescue.

The potential importance of mental impairment during hypothermia has not gone unrecognised, and has been experimentally studied by several investigators. Despite the apparent simplicity of the problem, no

clear answer has emerged to the question: "Does hypothermia impair mental abilities?" Anecdotal reports are clear in their descriptions of alteration in mental state or conscious level with severe cold. Experimental studies are of course limited by the ethics of cooling volunteers.

In the past two decades there have been several studies on this problem, and these are further detailed below. Generally, either no effect of cold was found, or else effects were found which were attributed to either distraction by cold, hand numbing by cold, or CO_2 build-up. Whether cold itself impaired mental processes directly could not be deduced, because the method of cooling did not separate muscular chilling and falls in deep body temperature. Or else data was analysed for groups of subjects, often where deep body temperature was unknown, or where results were pooled from subjects who became hypothermic and those who did not.

These experiments are described in detail below, partly because they are the only attempts to determine the effects of cold in this area, and partly because further investigation would have to avoid the same pitfalls. There is some evidence that cooling does not have to be profound to cause mental impairment: Complete amnesia for the last few minutes of 20-minute immersions in water at 5°C by two subjects was noted by Keatinge (1983). Their core temperatures were 34.2 and 35.1°C. In a short personal account Hamilton (1980) describes his astonishment at finding he was unable to subtract serial 7's when his deep body temperature fell to 34.5°C during cold water testing of helicopter survival suits. In a retrospective survey of 85 canoeists, Baker and Atha (1981) found that after capsize in water at 8°C, 53 reported disorientation, dizziness and visual problems. No less than 5 lost con-

sciousness. Bowen (1968) considered that cold was the major environmental stress affecting divers, and designed a series of experiments to test motor, mental and sensory functions during cold immersions. The conditions were arranged to simulate a genuine dive as closely as possible, and the sixteen sugjects wore neoprene wet suits, masks and scuba apparatus. Body temperatures were obtained by urine sampling immediately after immersion (average temperature 35.8°C). The dives took place in water at 8.3°C with similar dives at 16.7°C as controls. Grip strength was measured with a dynamometer, tactile sensitivity using a variation of 2 point discrimination, manual dexterity using a simple peg and ring and threaded (screw plate) system, with further tests of mental and physical function - group assembly of a pipe structure and a "tracking" test involving moving a peg along a twisting track by two remote controls. Purely mental abilities were tested by a series of simple 3-digit additions, a "symbol processing" task involving sequencing numbers and colours, and a "set exceptions" test consisting of selection of one of 5 numbers without a common denominator. Short-term memory was assessed by the ability to recall times set on a series of clock faces, where eight different faces were displayed on a board, inspection allowed for one minute and then the times on the faces reproduced by drawing a diagram thirty seconds later. Results confirmed losses of tactile sensitivity, grip strength and manual dexterity after exposure to cold. There was a significant slowing for psychomotor tests performed in colder water (30% slower for the screw plate test and 55% slower for the two-handed tracking test). Group pipe assembly was also slower. There was no decrement in accuracy of the tasks. Similarly, fewer of the simple arithmetic tests were attempted, and there was no loss of accuracy. Symbol processing and set exceptions did not show an effect of

cold exposure, but more problems were omitted in the latter test in the colder water. The clock test was less accurate in both water temperatures than on dry land.

Perhaps because of the desire to simulate a dive closely, the data presented has a limited potential for analysis. Firstly, the degree of cooling as measured by urine temperature is given as an average temperature, and then for only seven of the sixteen divers, the others having been discarded because of spillage, contamination, or inability to micturate immediately post-dive. Thus it is impossible to link observed decrements with alteration in core temperature. It is not possible to separate the effects of cold on manual dexterity and mental processes because the results of the tasks were manually communicated, either by writing with a wax pencil, or moving a ring, pipe, or screw. The authors concluded that there were some demonstrable effects of cold, but that these were likely to be mediated by the distraction effects of cold water, and reduced manual dexterity by chilling. Because of the probable mild degree of cooling achieved, no gross cognitive changes were seen, but it is interesting that the authors recorded complaints of lapses of short term memory in divers during the two hours following the dive. It is possible that the subjects were experiencing an "afterdrop" in deep body temperature (see Chapter Five) at this time, because rewarming had been by drying and re-dressing rather than the hot bath method.

Stang and Weiner (1970) measured the performance of 12 divers in water at 10°C, 15°C and 21°C. The temperature data was again presented as an average for the group and analysis thus limited. Each performed manual dexterity tasks, choice reaction tests (switching off a randomly presented light), and mental arithmetic. A slowing of the choice reac-

tion time and manual dexterity was found in the coldest water. It was likely that the divers cooled appreciably, since the immersions were of 90 minutes' duration, but temperatures were measured in only seven divers. Furthermore, the method chosen was oral temperature via a breathing mouthpiece and is likely to have been very unreliable (Keatinge and Sloan, 1975).

Vaughan (1975) was aware of the difficulties in attributing aspects of performance in the cold to peripheral cooling or core cooling, and measured rectal temperatures of 6 divers controlling the depth of a submersible over 4 hours in 16.5°C water. He found depth control was not different from hour to hour, or from training in warmer water. Rectal temperatures were presented as an average fall of 0.7°C, with the largest being a fall of 1.83°C. Vaughan (Vaughan and Mavor, 1972) extended his work, considering that the previous submersible trials had failed to show an effect of cold because cooling had been insufficient, and the previous task had been too easy. Continuous monitoring of submersible heading was added to the depth maintenance task. Exposures were longer (up to 6 hours), and twelve divers were used. Core temperature was measured with both rectal probes and temperature sensitive radio-pills. The standard deviation of frequent measurements of depth and heading were used as a quantitative measurement of diver performance. Significant error was detected compared with controls after 2 hours' exposure for heading but not for depth. Five of the divers reported "confusion", "mental difficulties" and "memory lapses" but these were not further explained.

Although Vaughan's work gives a picture of likely performance under field conditions, the contributions of boredom, peripheral distraction and falling core temperatures were not separated. The potential accur-

acy of temperature measurement by rectal probe and radio pills was counterbalanced by the presentation of data in a group form (mean ± SD changes in core temperature). It was noted that there was little difference in temperatures between the 4- and 6-hour dives. This probably indicated that thermal balance had been achieved. The mean fall in temperature for the 6-hour dive was 1.2°C and it is likely that some divers were not cold enough to manifest central effects on performance.

Baddeley et al (1975) exhaustively investigated the same problem. Initial studies were made on 14 divers wearing wetsuits, and immersed in 25.6°C and 4.4°C water for an hour. Temperatures were measured with rectal thermistors, and results pooled, with an average fall of 1.2°C during the colder immersion. Various tests of mental performance were conducted. A series of reasoning tests, the answer to each of which was true or false (eg. AB, A comes after B) (Baddeley, 1968), a visual vigilance test (verbal response to random illumination of a light source within the facemask), a pipe assembly task, and a memory test, where a paragraph with details about sunken ships was memorised when cold and recall later tested.

There was no detectable effect on speed or accuracy of the AB reasoning test either at the start or end of immersions. Memory did appear to suffer in the cold, but the effect was not marked. Vigilance was unaffected. The pipe assembly task, involving physical manipulation, took longer.

Because only minor effects could be detected, the work was extended by modifiying the tests, in an attempt both to reduce the effects caused by muscular chilling, and by complicating the tests of mental performance (Davis, Baddeley and Hancock, 1975). Fifteen divers were tested

during 40-minute dives in water at 20°C and 5°C. The average drop in rectal temperature was 1.4°C during the 5°C dive.

An arithmetic test addition was simplified from Bowen's 1968 method by reducing the amount of writing required. The AB reasoning test was changed to a more complex form where two statements were followed by four conclusions, one of which was correct. A digit-span memory test involved the simple repetition of a series of spoken digits, scored either as correct or incorrect. A more complicated test of memory was also used, where the subjects were afterwards presented with a list containing words he had learnt when cold, and asked to tick those he recognised, as well as writing out a list of words he could spontaneously remember. Finally, manual dexterity in the cold was tested using a screw-plate system.

More definite results were obtained from Baddeley's second study. The speed of arithmetical calculation slowed. Each sum taking an average 6 seconds in the warmer water and 7.2 seconds in the cold, without an increase in errors. There was no change in the reasoning tests at the start of immersions or at the end of the warm dives, but less problems were attempted during the cold dives. The percentage of errors on this test was variable, but not significantly different in the cold. The digit span memory test was the same under all conditions, while the more complicated memory test scores were 3 ± 19 words learned in the cold and 9 ± 38 in the warm (mean $\pm$ standard error). This latter dramatic difference was also statistically significant.

The test of manual dexterity demonstrated slowing in both warm and cold water compared with land, but the slowing was not more marked in the cold.

The authors concluded that their most important finding was the memory deficit that ocurred in cold water, over 70% worse than performance on dry land. Performance in other aspects of cognitive function also seemed reduced in the cold, with a reduction in speed of reasoning and calculation, rather than any change in accuracy. Although there was an experimental correlation between fall in rectal temperature and speed of reasoning, the authors were hesitant to attach much importance to this. This was because speed of reasoning was also slowed to a similar degree at the start of the cold dives, and thus the distracting effect of cold water seemed as important as, if not actually the whole explanation for the observed slowing.

Finally, Biersner (1976) measured the results of a ring and peg and colour coding test of reasoning in 4 divers breathing helium-oxygen in a hyperbaric chamber. Water temperatures were 32°C and 8°C at atmospheric pressure and 183 m. Body temperatures were not measured. Significant slowing was seen with all water environments compared with the dry. At the end of deep cold dives there was further slowing not seen with the dry, warm, or shallow cold environments. Slowing was thought to be due to a distraction effect by cold, and in particular slowing in deep water was considered to be due to CO_2 buildup in the breathing apparatus. This was chiefly because CO_2 had been found in earlier work to be higher in similar conditions, and that CO_2 had been previously shown to impair motor performance. In the experimental series involving 4 divers, however, CO_2 was not measured.

The cited work suggests that there are four potential effects of cold on diver performance. An initial impairment due to thermal and other environmental distractions, a peripheral effect due to muscle

chilling in the limbs, an effect due to increased CO2 in breathing gas, and a central or cognitive effect.

Evidence for impairment of manual dexterity in the cold is good (Fox, 1967; Gaydos, 1958; Gaydos and Dusek, 1958; Teichner, 1958) and this finding is not really surprising, considering the reduction in skin and muscle blood flow by cold, and the temperature-sensitive nature of neuromuscular coordination.

Evidence for an immediate effect by distraction also seems sound (Davis et al, 1972), and is again not surprising, as sudden cooling causes hyperventilation and reflex bradycardia (Keatinge and Evans, 1961).

CO2 buildup cannot be the explanation for cognitive changes occurring where no breathing apparatus is worn, although this may well be a factor under hyperbaric conditions.

Despite careful investigation, the effect of central cooling on cognitive processes remains unclear. There are indications that memory suffers and thought slows. At what brain temperature does this occur? Is there a threshold or a gradual reduction in abilities with cooling? How severe does the cognitive defect become? Are different mental processes affected in different ways?

The design of previous studies was such that it is not possible to separate the effects of a cold skin or cold muscles from the effects of a cold brain.

In most, the deep body temperature was not correlated with performance except as a group. This is an important point, and vital to a correct interpretation of the results. Thermal protective gear was worn in all previous studies, and it is likely that some subjects in the cold exposed groups did not experience deep body cooling at all. This may

have been satisfactory if subjects had been separated into those who showed such cooling and those who did not, but in no case was this done.

Thus the main drive of the next series of experiments was careful design to allow separation of factors that may impair mental performance.

REFERENCES

Baddeley AD (1968). A 3-minute reasoning test based on grammatical transformation. Psychon Sci 10, 341-342.

Baddeley AD, Cuccaro WJ, Egstrom GH, Weltman G, Willis MA (1975). Cognitive efficiency of divers working in cold water. Hum Factors 17, 446-454.

Baker S, Atha J (1981). Canoeists disorientation following immersion. Brit J Sports Med 15, 111-115.

Biersner RJ (1976). Motor and cognitive effects of cold water immersion under hyperbaric conditions. Hum Factors 18, 299-304.

Bowen HM (1968). Diver performance and the effects of cold. Hum Factors 10, 445-464.

Currie J (1797). Medical reports on the effects of water, cold and warm. Cadell and Davies, London, pp.20-45 (Appendix II).

Davis FM, Baddeley AD, Hancock TR (1975). Diver performance: The effect of cold. Undersea Biomed Res 2, 195-213.

Davis FM, Chorlies AR, Sammarez R, Muller V (1972). Some physiological responses to the stress of aqualung diving. Aerosp Med 43, 1083-1088.

Fox WF (1967). Human performances in the cold. Hum Factors 9, 203-220.

Gaydos HF (1958). Effect on complex manual performance of cooling the body while maintaining the hands at normal temperatures. J Appl Physiol 12, 373-376.

Gaydos HF, Dusek ER (1958). Effects of localised hand cooling versus total body cooling on manual performance. J Appl Physiol 12, 377-380.

Hamilton SJC (1980). Hypothermia and unawareness of mental impairment. Brit Med J 565, 23rd Feb.

Keatinge WR (1983). Quoted in: "Impaired memory registration and speed of reasoning caused by low body temperature." Coleshaw SRK, van Someren RNM, Wolff AH, Davis HM, Keatinge WR. J Appl Physiol 55, 27-31.

Keatinge WR, Evans M (1961). The respiratory and cardiovascular response to immersion in cold and warm water. Q J Exptl Physiol 46, 83-94.

Keatinge WR, Sloan REG (1975). Deep-body temperature from aural canal with servocontrolled heating to outer ear. J Appl Physiol 38, 919-921.

Lubbock B (1932). The Nitrate Clippers, p.39. Brown, Son and Ferguson, Glasgow.

Stang PR, Weiner EL (1970). Diver performance in cold water. Hum Factors 12, 391-399.

Teichner WM (1958). Reaction time in the cold. J Appl Psychol 11, 333-338.

Vaughan WS Jnr (1975). Diver temperature and performance changes during long-duration cold exposure. Undersea Biomed Res 2, 75-88.

Vaughan WS Jnr, Mavor AS (1972). Diver performance in controlling a wet submersible during four-hour exposures to cold water. Hum Factors 14, 173-180.

CHAPTER FIVE

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THE EFFECTS OF MILD HYPOTHERMIA ON MEMORY

An unusual physiological state exists during hot-water rewarming following cold immersion. Shivering, and the sensation of cold cease after one to two minutes in a hot bath of around 41°C. However, deep body temperature fails to rise, and usually falls further before normal body temperature is regained some 20-30 minutes later.

The magnitude of this further fall in temperature during initial rewarming is substantial and may be 1-2°C. In 1797, Currie found he could clearly record the phenomenon using an oral mercury thermometer. Termed 'afterdrop', the precise mechanism is not fully elucidated. The rate of afterdrop, and probably the degree, are related to the physical rather than physiological thermal gradients within the body prior to removal from cold water, with a lesser contribution from the cessation of extra heat production by shivering, and circulatory readjustments (Behnke and Yaxlow, 1950; Collins, Easton and Exton-Smith, 1982; Golden and Hervey, 1981). As a general rule, the afterdrop is more severe and prolonged with longer and colder periods of cold immersion.

The occurrence of this phenomenon provides a valuable tool for disassociating the effects of skin cooling and deep body cooling. Thus, the main hypothesis of most previous authors can be critically analysed, i.e. that effects of cold on cognitive processes are due to distraction by skin cooling. A subject can be cooled in a cold immersion tank for a period of time, and subsequently rewarmed in a hot bath. During the rewarming, the skin rapidly becomes comfortable and shivering ceases. However, the temperature of the brain and deep tissues will remain low or fall further for up to 20 minutes after entering the hot bath. This provides ample time for simple psychological testing without the distracting effect of environmental cold. The value of the experimental results then depends on the quality of the control work for other facets

of the experimental conditions, such as opportunity for learning, the immediate experimental environment, the replicability and relevance of the psychological tests chosen, and subject grouping and numbers.

After consideration, a simple experimental format was devised, using standard paragraphs for memorisation.

METHODS

Thirty-three volunteers were used in the memory experiments. They were medical students, hospital personnel and members of a local amateur sub-aqua club. All were medically examined prior to immersion.

The basis of the experiment was to randomly assign the subjects to either of two groups and cool and rewarm each group in an identical manner. During the rewarming, one group memorised a standard paragraph, while the other recalled a standard paragraph which had been learned prior to immersion. Effects of cold on information storage and on retrieval could therefore be separated. Subjects were immersed individually, and on experimental days there was usually one, or only occasionally two, separate immersions. Subject instrumentation was the zero gradient aural thermometer and electrocardiogram leads.

Immersions took place in the laboratory and each subject spent an hour in water at 15°C within the immersion tank of 4,000 L of thermostatically-controlled water stirred by ducted compressed air. At the end of 60 minutes, the subject climbed out of the tank and walked to the rewarming bath, situated 5 feet from the tank. The rewarming bath had been previously filled with water at 41°C and the subject remained there until he had regained his pre-immersion aural temperature.

Two aspects of memory were tested. Firstly, the ability to recall previously learned information while in a hypothermic state, and second-

ly the ability to retain information learned while hypothermic. For the purposes of the experiment, the latter was termed 'registration' and the former 'recall'. Two passages of the type devised by Friedman and Grietzer (1972) were used. This was to enable some kind of comparison to be made with previous use of the passage by Baddeley et al (1975).

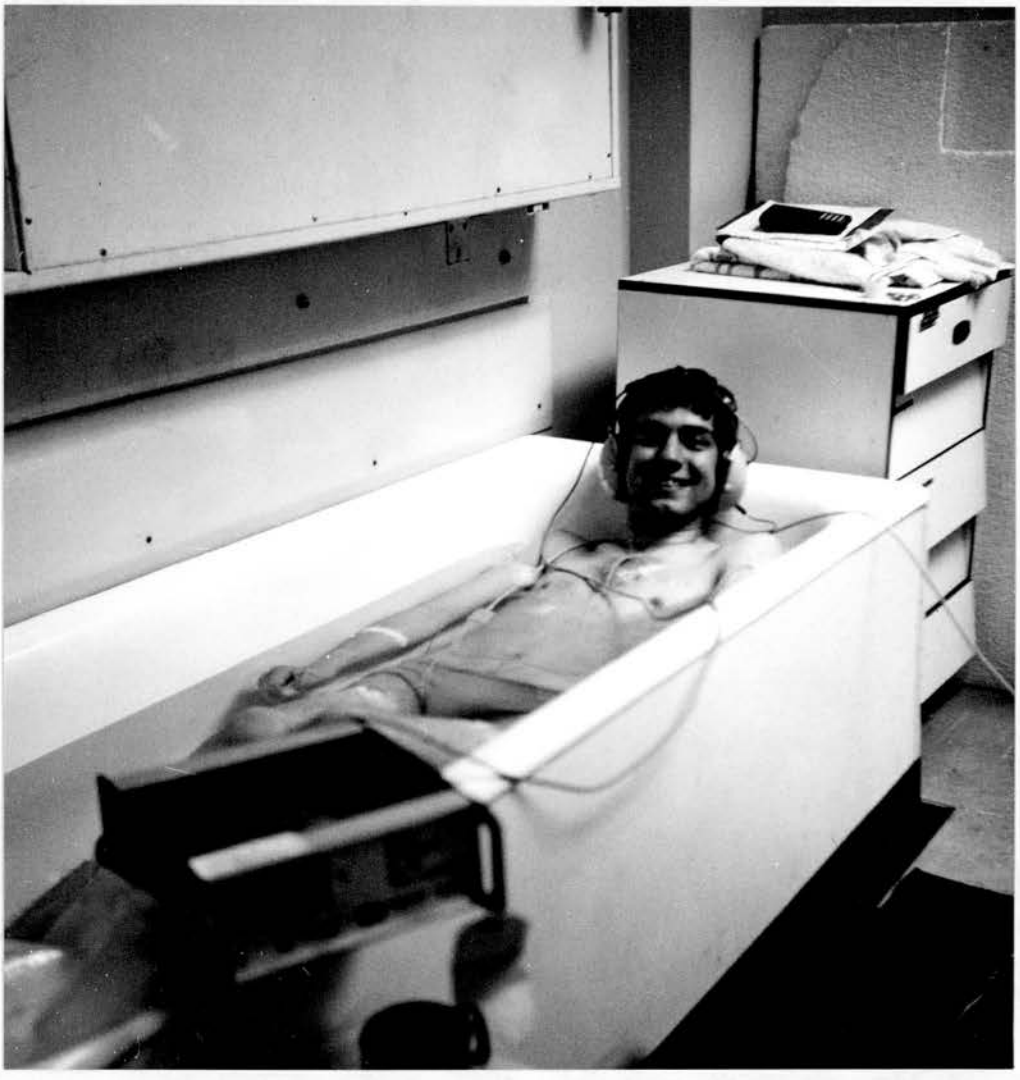
MEMORY PASSAGE I

> Around a small island there are a number of boats that have been wrecked. There are three which are well known to divers. The first is a fishing-boat called The Lady Lucy which lies in 3 metres of water amongst kelp; the only danger is the number of poisonous fish. The second, which is surrounded by sharks, is a battle-ship, HMS Intrepid, which sank in 20 metres of water and lies on a shingly bottom. The third is a coaster, called The Mermaid, which lies on a rocky bottom in 50 metres of water, but is now only a tangle of metal with very sharp edges.

MEMORY PASSAGE II

> Around a small island there are a number of boats which have been wrecked. Three of them are well known to divers. The first is a liner called The Olympus which lies in 24 metres of water in a gulley through which there are very fast currents. The second, which is enclosed in low visibility, is a yacht, The Nipper, which sank in 11 metres of water and lies on a sandy bottom. The third is a cabin cruiser, called The Limpet, which lies at the foot of some cliffs in 5 metres of water and is subjected to dangerous swell.

Each passage was typed on waterproof card nine inches square and was held by the subject for 2½ minutes while learning. He was informed when one minute remained, in order to maximise use of available time, in the manner used by Friedman and Grietzer (1972).



Rewarming bath.

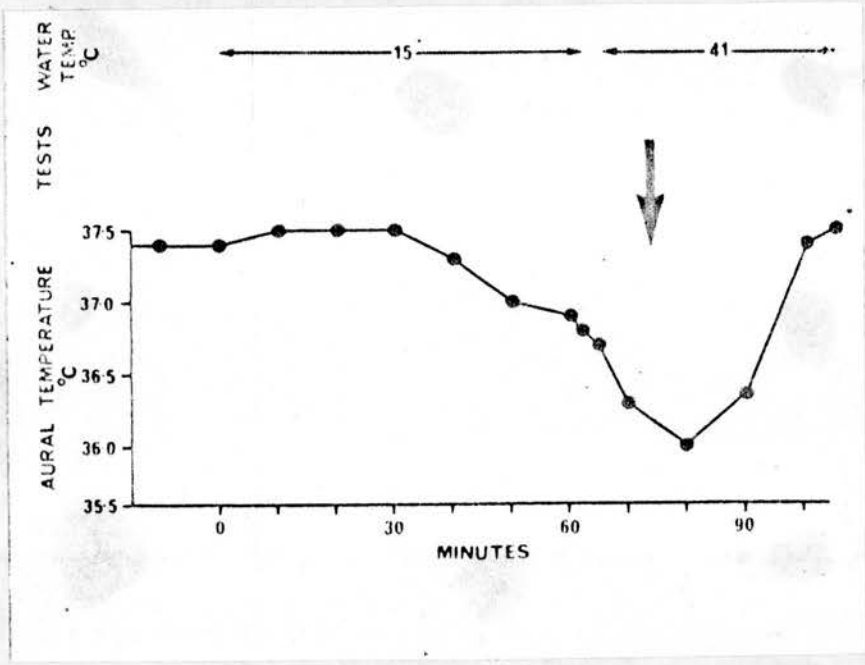


FIGURE 1: The aural temperature of one subject at various times during 15°C immersion and subsequent rewarming on 41°C. The arrow indicates the memorisation period.

MEMORY REGISTRATION

Fourteen volunteers were used. All simultaneously participated in other cognitive experiments (Chapter Six) but not in other tests of memory.

Subjects were handed the passage to learn 2-3 minutes after entering the warm bath, when they felt comfortable and had ceased shivering (see Fig. 1 for deep body temperature at this time). They then learned the passage, and 60 minutes later were asked to recall as much as they could remember, by which time they were fully rewarmed, dressed and seated in the immersion laboratory. The information recalled was written down verbatim by an observer. No time limit was placed on this, although in practice this took no longer than three minutes.

SCORING

The simplest method was simply a count of the number of completely correct facts. Maximum score was 15. It was thought that more subtlety could be achieved by scoring the kinds of error, such as omissions, fabrications, inaccuracies (e.g. "Lucky Lady" instead of "Lady Lucy") or transpositions (e.g. "the Lady Lucy lies in 20 m of water" instead of "3 m of water"). Minor errors were not penalised (e.g. "feet" instead of "metres"). Scores for each type of error were one mark for a transposition or a completely correct statement, half a mark for an inaccurate statement, and no marks for an omission or a fabrication.

CONTROLS

The similar alternative passage was used. During a pre-immersion acclimatisation visit to the laboratory 1-7 days beforehand, each sub-

ject was asked to memorise the control passage in 2½ minutes and recall it 60 minutes later. The facts recalled were scored in the same manner as the experimental passage.

The choice of Passage I or II as experimental or control test was a random one, with 7 subjects using Passage I as a control and 7 subjects using it during the experiment.

RESULTS

MEMORY REGISTRATION

Controls

Each subject acted as his own control. There was no correlation between control and experimental scores, indicating no effect of familiarity or learning by the experimental procedure. There was also no correlation between body temperature at the time the control passage was learned and control score, suggesting that within the normal temperature range, the difference in scores was not due to body temperature variation ($R = 0.17$, and $R = 0.09$).

Finally, there was no relationship between the control memory score and body temperature under experimental conditions, indicating that the strong relationship (see next paragraph) of subnormal body temperatures to memory deficit is due to cooling alone ($r = 0.19$).

Experiment

TYPES OF ERROR

In Table 1, the types of error made when recalling the passage are tabulated against the aural temperature at the time of registration of

memories, and in Table 5 the same data is grouped together for statistical analysis. It is clear that the omission of data is by far the most common error and that this is related to the fall in aural temperature.

EFFECTS OF TEMPERATURE

A regression line was drawn of aural temperature and each subject's experimental score deficit compared with his control score (Fig. 2). This demonstrates a clear relationship between fall in aural temperature and reduction in accuracy of registration of memories ($r = 0.77$; $p < 0.002$). There is a steady decrease in experimental score with lower body temperature and no evidence of a 'threshold' temperature below which memory suddenly worsens.

Memory recall

Nineteen subjects took part in experiments to determine the ability to recall information when cerebral temperature was low. All were immersed for 60 minutes in 15°C water and then transferred to the hot bath. They wore an aural thermometer and electrocardiograph leads and both were continuously monitored. Once lying in the hot bath, and comfortable, they were asked to recall as many facts as possible from the memory passage which they had learned immediately prior to immersion. At the time of learning, they were instrumented and sitting in the laboratory. Each was given the nine inch square waterproof card to hold and memorised the printed paragraph over 2½ minutes. A reminder was given when a minute remained. Scoring methods were the same as the previous 'registration' experiments.

TABLE 1: Types of error in remembering passage, and aural temperature at the time the passage was learned.

SUBJECT	AURAL TEMP. (°C)	OMISSIONS	INACCURACIES	FABRICATIONS	TRANSFERS
1	34.2	5	2	2	-1
2	34.3	12	-3	0	0
3	34.6	7	-2	-1	1
4	34.8	10	1	0	-1
5	35.5	9	0	0	1
6	35.5	0	2	2	-1
7	35.8	-2	0	0	0
8	36.0	1	0	1	0
9	36.0	1	0	2	4
10	36.1	4	1	0	0
11	36.1	3	1	-1	0
12	36.5	2	-1	0	1
13	36.5	0	1	-1	0
14	37.1	2	-1	0	0

Columns state the difference in types of error between control and experimental passage. A negative sign indicates fewer errors under experimental conditions than in control conditions.

Group analysis (related t-test). Mean difference in scores 3.9 (±1.1) facts, $P < 0.01$.

	SCORE (mean ± SE)	TEMPERATURE (mean ± SE) °C
Control	12.5 ± 0.5	37.3 ± 0.1
Experimental	8.6 ± 1.1	35.6 ± 0.2

TABLE 3: Memory Recall

	GROUP ANALYSIS	
	SCORE (mean ± SE)	TEMPERATURE (mean ± SE °C)
Control	12.0 ± 0.7	37.2 ± 0.6
Experimental	11.3 ± 0.6	36 ± 0.2

Using the related t-test mean score difference is 0.44 and P > 0.05.

TABLE 4: Control data for memory 'recall' scores

	SCORE (mean ± SE)	
	PASSAGE I	PASSAGE II
Control pre-experiment	11.2 ± 1.1	11.8 ± 0.9
Control post-experiment	11.4 ± 0.7	11.7 ± 1.0

(a) No significant correlation between control body temperature and score ($r = -0.3$)

(b) No significant correlation between control score and experimental body temperature ($r = 0.02$)

(c) The two memory scores were related ($r = 0.71$, $P < 0.001$)

(d) Check for effects of learning control before or after experimental memory test - no effect

TABLE 5: A comparison of types of errors in the two memory tests.

REGISTRATION					
		OMISSIONS	INACCURACIES	FABRICATIONS	TRANSFERS
CONTROL	Mean	2.64	1.07	0.50	0.36
	SD	3.03	1.14	0.65	0.74
	SE	0.81	0.31	0.17	0.20
EXPERIMENT	Mean	4.93	1.21	0.79	
	SD	4.30	1.25	1.19	1.08
	SE	1.15	0.33	0.32	0.29
RECALL					
		OMISSIONS	INACCURACIES	FABRICATIONS	TRANSFERS
CONTROL	Mean	2.32	1.21	0.32	0.53
	SD	2.63	1.47	0.48	1.07
	SE	0.60	0.34	0.11	0.25
EXPERIMENT	Mean	2.89	0.84	0.21	0.37
	SD	2.56	0.90	0.42	0.60
	SE	0.59	0.21	0.10	0.14

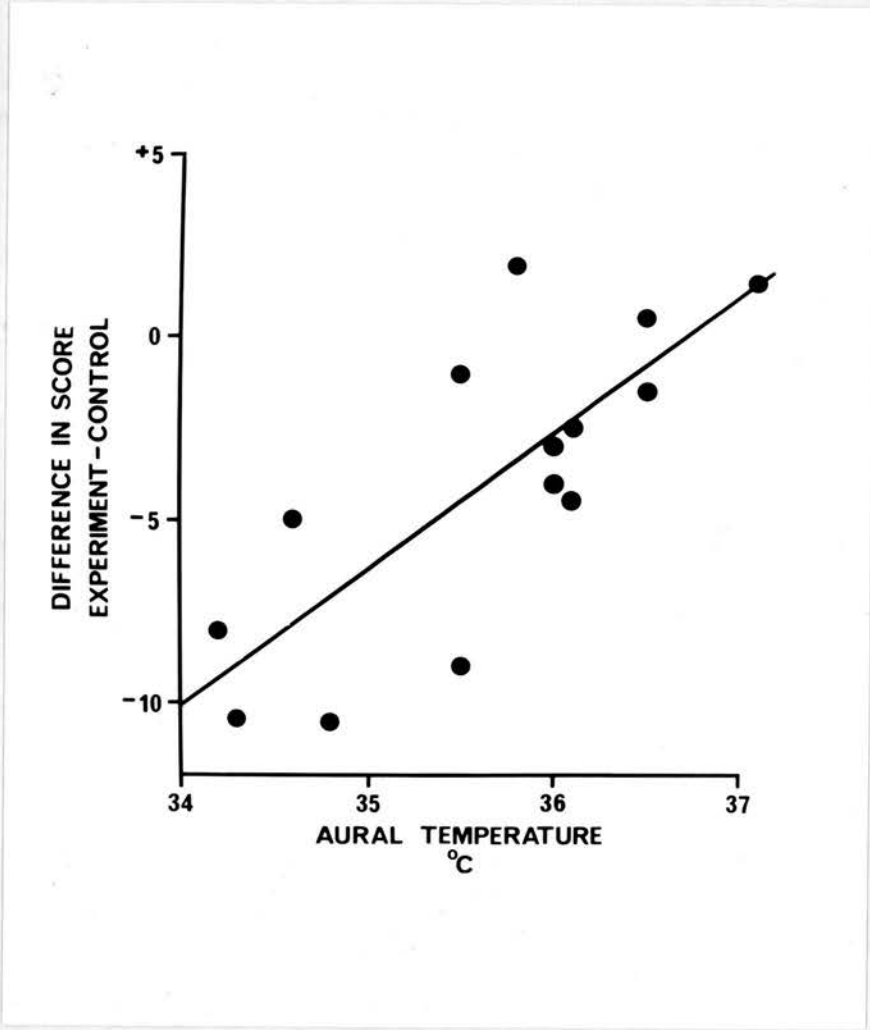


FIGURE 2: Memory registration at reduced core temperature. Facts subsequently recalled at normal body temperatures in relation to body temperatures present when they were memorised after end of cold immersions. Number recalled is given as difference from number recalled by same subject in prior control test in which both memorisation and recall took place at normal body temperature (mean no. of facts recalled in controls, 12.5). Decrement of facts registered = 3.7 (36.7 - body temperature, °C).

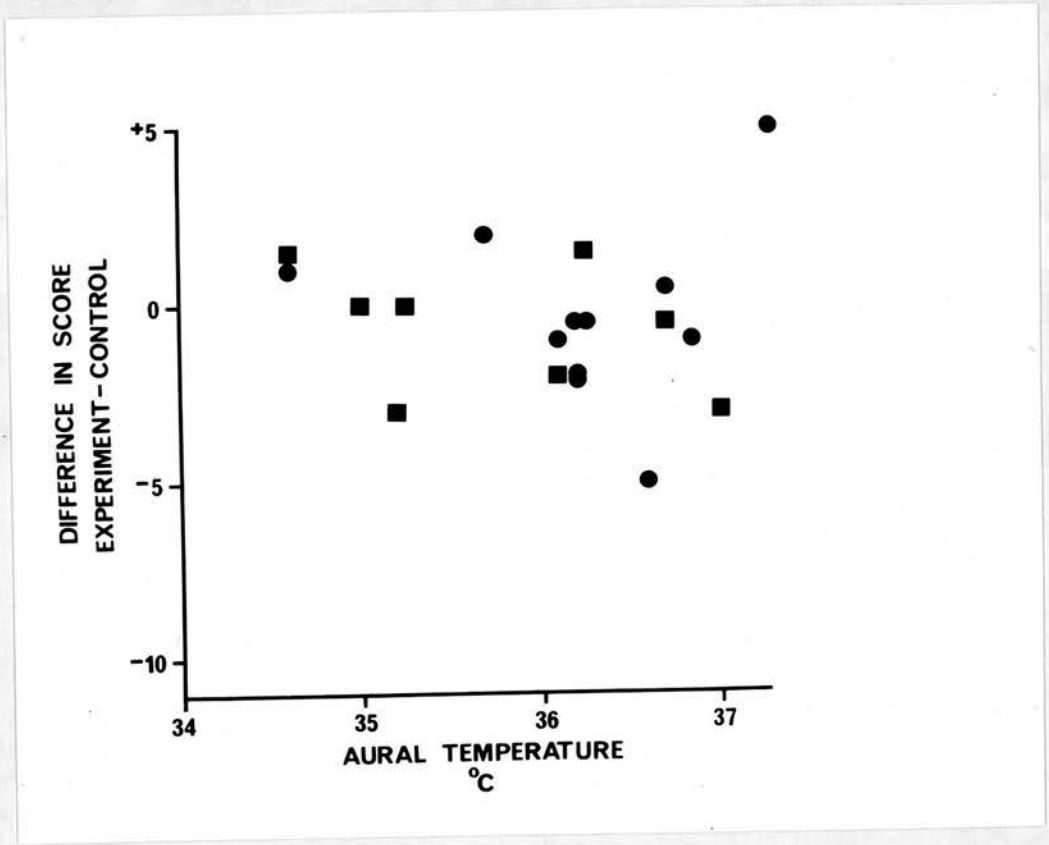


FIGURE 3: Facts recalled in relation to body temperatures at time of recall after end of cold immersions with facts previously memorised at normal body temperature. Number recalled is given as difference from number recalled by same subject in control tests, given either before (●) or after (■) immersion, in which both memorisation and recall took place at normal body temperature (mean no. of facts recalled in controls, 12.0).

Subjects learned the alternative, randomly-assigned, passage either up to a week before or after the experimental immersion and scoring was carried out in a similar manner.

MEMORY RECALL RESULTS

There was no alteration in either the number or type of error compared with controls, when compared with aural temperature at the time of recall ($r = -0.3$) (see Fig. 3 and Tables 3 and 4). The degree of subject cooling was similar to that in the 'registration' experiments, with three subjects between 34°C and 35°C (cf four) and five subjects between 35°C and 36°C (cf three).

Controls (see Table 4)

There was a significant relationship ($r = 0.71$, $p < 0.001$) between the control and 'recall' scores. This is supporting evidence that temperature had no effect on memory recall, the scores remaining compatible with the memory abilities of individuals at normal temperature.

As in the 'registration' experiments, there was no correlation between control body temperature and score, or between control score and experimental aural temperature.

In addition, a comparison was made between the scores of subjects performing the control test before the experiment and those afterwards, but no bias was uncovered.

DISCUSSION

Because each subject acted as his own control, the analysis was uncomplicated and did not require large numbers of subjects.

There was no detectable influence of a learning process between memorising the first and second passages, since there was no difference in scores within the sub-group who learned the passage prior to immersion whether the control passage was learned before or after this. Both passages were of equal difficulty, in that scores were the same for both groups when learned as controls in the warm.

Care was taken to minimise the phenomenon of state-dependent recall, i.e. memory recall is better in the same surroundings as those during memorisation (Greenspan and Ranyard, 1957). The memory passage in the present experiments was memorised and recalled in the immersion laboratory for both experimental and control episodes, the difference being that the subjects were fully clothed for the control memorisation. However, the difference in clothing cannot account for the memory decrements found, since similar decrements should then be found in all subjects. One may argue that the low body temperature acted in a similar maner to the differences in clothing, but the counter-argument is the same, i.e. all subjects should have been equally affected.

The conclusion is inescapable that a low-brain temperature itself hinders the ability to learn new material, while that learned at normal temperatures, in contrast, can be recalled at low body temperatures just as well as in the warm.

Hebb (1949) has proposed that the physical basis of learning involves two types or phases of learning process: a short-term 'consolidation' of recent experience, and a long-term permanent storage of the memory trace. The evidence on which this is based is experiments which show that drastic alterations in the state of brain activity interfere selectively with recently-learned responses. Traumatic retrograde amnesia following head injury, i.e. where events immediately prior to

the incident are forgotten, is a common human experience (Russell and Natham, 1946). Duncan (1949) demonstrated that if an electroconvulsive shock was administered to rats who had learned to avoid a shock to the feet in a shuttle box, there was a deleterious response in the ability to learn which was more marked the closer was the electroconvulsive shock to the learning trial. In a variety of subsequent animal experiments, epileptic convulsions (Pearlman, Sharpless and Jarvik, 1961), hypoxia (Thompson and Pryer, 1956) and depressant drugs (Steinberg and Summerfield, 1957) were shown to have similar effects, with the stimulus-insult interval necessary ranging from two minutes to an hour.

The length of time necessary to establish a memory has been hypothesised to be due to 'reverberating neural circuits' (Hebb, 1949), in which activity cycles in closed loops around chains of neurones, representing initial storage of the memory trace. During the half-hour or so of reverberation, more permanent memory traces would then be laid down. Procedures that interfere with electrocortical activity should have a progressively greater effect on learning as they are applied closer in time to the learning trials. The precise nature of the physiological underlying process is unclear (John and Schwartz, 1978).

The results of the present experiments are compatible with a consolidation hypothesis: the time between learning and recall was 60-90 minutes, which was presumably enough time for a permanent memory trace to have been established. If no stress due to hypothermia occurred, this memory remained intact. However, it should be borne in mind that the circumstances were not quite the same as in animal experiments or traumatic amnesia, because learning took place during the time the brain was cool rather than an insult occurring at a time following the initiation of the learning process. Patients kept for up to three weeks

at body temperatures of 30-31°C for malignant astrocytomas (Clarke and Edholm, 1984) recovered consciousness at these temperatures and appeared to behave normally, but on subsequent rewarming had no memory of the time they had spent at low body temperatures. Similarly, low-dose anaesthesia, in which the ability to read and communicate is unimpaired, seems to produce equivalent effects to hypothermia on memory; the acquisition of a learned process rather than its demonstration is affected, without great effect on recall of long-term memory (Adam, 1973). This implies that cognition may be unaffected but memory impaired by more subtle insults.

As far as hypothermia is involved, it may well be that verbal memory is affected before non-verbal memory, such as during light anaesthesia. If this is true, then during diving with subsequent mild hypothermia, tasks involving speech (e.g. remembering an instruction "you have fifteen minutes left") may be preferentially forgotten to those involving spatial or organisational memory, such as re-tracing a route out of a wreck.

The practical importance of the finding of memory deficit caused by mild hypothermia is clear. A diver will be able to recall pre-dive instructions but may have difficulty reporting after a dive. The evidence provided here is the first objective rather than anecdotal report describing the type of memory impairment due to mild hypothermia.

REFERENCES

ADAM N (1973). Effects of general anaesthetics on memory functions in man. J Comp and Physiol Psychol 83, 291-305.

BADDELEY AD, CUCCARO WJ, EGSTROM GH, WELTMAN G, WILLIS MA (1975). Cognitive efficiency of divers working in cold water. Human Factors 17, 446-454.

BEHNKE AR, YAZLOW CP (1950). Response of man to chilling and rewarming. J Appl Physiol 3, 591-596.

CLARKE RP, EDHOLM OG (1984). Man and his thermal environment. Edward Arnold, p.160.

COLLINS KJ, EASTON JC, EXTON-SMITH AN (1982). Body temperature after-drop: a physical or physiological phenomenon? J Physiol 328, 72P.

CURRIE J (1797). Medical reports on the effects of water, cold and warm. Cadell and Davies, London.

DUNCAN CP (1949). The retroactive effect of electroshock on learning. J Comp and Physiol Psychol 42, 32-34.

FRIEDMAN MP, GRIETZER FL (1972). Organisation and study time in learning from reading. J Educat Psychol 63, 609-616.

GOLDEN F St C, HERVEY GR (1981). The 'after drop' and death after rescue from immersion in cold water. In: Hypothermia Ashore and Afloat. Proceedings of the 3rd International Action for Disaster Conference. University Press, Aberdeen.

GREENSPAN J, RANYARD R (1957). Stimulus conditions and retroactive inhibition. J Exptl Psychol 53, 55-59.

HEBB DO (1949). The Organisation of Behaviour. Wiley, New York.

JOHN ER, SCHWARTZ EL (1978). The neurophysiology of information processing and cognition. Ann Rev Psychol 29, 1-29.

PEARLMANN CA, SHARPLESS CK, JARVIK ME (1961). Retrograde amnesia produced by anaesthetic and convulsant agents. J Comp and Physiol Psychol 54, 109-112.

RUSSELL WR, NATHAM PW (1946). Traumatic amnesia. Brain 69, 280-300.

STEINBERG H, SUMMERFIELD A (1957). Influence of a depressant drug on acquisition in rote learning. Q J Exptl Psychol 9, 138.

THOMPSON R, PRYER RS (1956). The effect of anoxia on the retention of a discrimination habit. J Comp and Physiol Psychol 49, 297-300.

CHAPTER SIX

—ooOoo—

CALCULATION AND REASONING TESTS AT REDUCED DEEP BODY TEMPERATURE

By exploiting the 'afterdrop' phenomenon, where the skin is warm but deep body temperature continues to fall for 20-30 minutes on rewarming after a cold immersion, changes in reasoning ability due to a low brain temperature were demonstrated.

Muscle cooling impairs manual dexterity (Clarke, Hellon and Lind, 1958), and the forearms and hands were maintained at a constant 29°C by using the hand and foot boxes described in Chapter Three, so that the results of whole body cooling in water at 15°C and 29°C could be compared.

SELECTION OF SUITABLE COGNITIVE TESTS

There were three main points to be taken into account. Firstly, the tests chosen should be comparable with those described by other authors. Secondly, there should be a minimum of limb movement involved in responses, so that any effect seen could be ascribed to mental processes. This excluded pipe puzzles, ring and pag tests and tracking tests. Thirdly, the tests should be simple and interpretable. For example, tests of vigilance and time estimation, although easy to perform, are not easy to interpret in the light of control processes or relevance to diving. Three types of mental test were chosen. Simple mathematical tests were appropriate, because calculation of numbers involving depth and times are a part of daily routine. Direct comparisons could also be made with the work of previous authors in the field (Bowen, 1968; Stang and Weiner, 1970; Davis, Baddeley and Hancock, 1975). The scoring of mental arithmetic is easy since the answers are either correct or incorrect. These tests involve short-term memory and a degree of intellectual ability.

Single-digit addition and double-digit addition tests were employed and both were similar to those used by previous authors. For the single-digit test, five digits were projected horizontally on a slide. For the double-digit tests, four double digits were presented horizontally

e.g. $7 + 6 + 8 + 9 + 5 =$

$72 + 21 + 94 + 55 =$

A third type of test was also chosen. This was the 'A-B' reasoning test devised by Baddeley (1968, and Appendix). This test had been devised as an indicator of 'higher functions' which was rapid and easy to perform. The score correlates well with intelligence quotient, and is reliable, with little practice effect on score. It had been used in stress studies of nitrogen narcosis, and in other tests, such as white noise interruption or car driving, showed demonstrable decreases in score. Baddeley had also used the test during an examination of the effects of cold on divers, and this made the test particularly attractive. Each reasoning problem consisted of a statement about the orders of the letters A and B, followed by either AB or BA. The answer required was either true or false. An example is printed below.

A does not precede B; AB

METHODS

Six racks of slides were made up. Each contained 10 slides with single-digit mathematical problems, 6 with double-digit arithmetical problems, and 8 AB reasoning problems, in that order. Each of the 144 slides was unique. The racks were always presented in the same

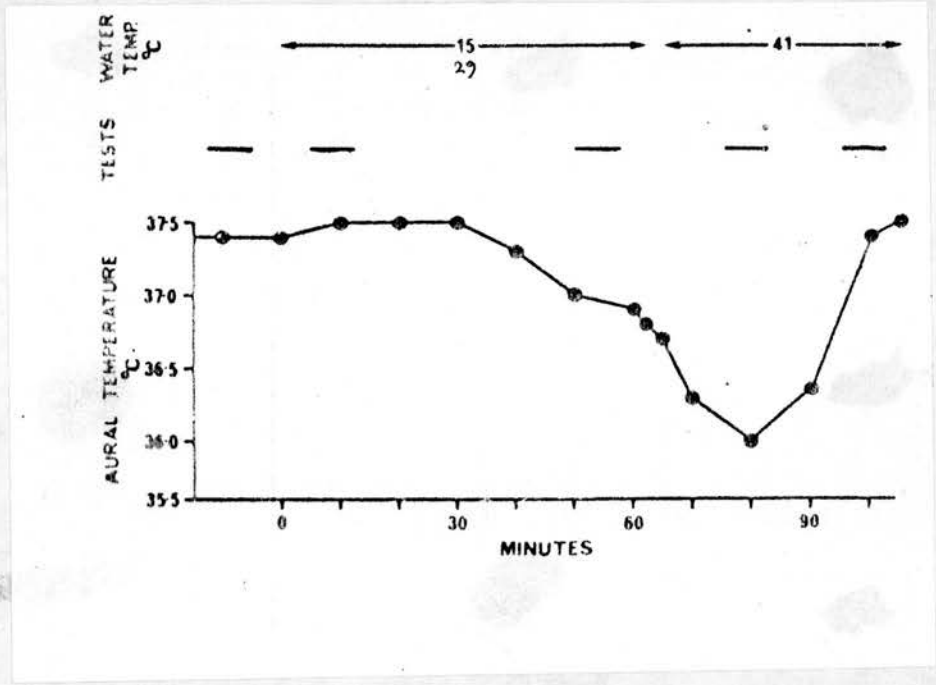


FIGURE 1: Timing of tests before and during cold and warm immersion with change in core temperature of one subject _____, calculation and reasoning tests.

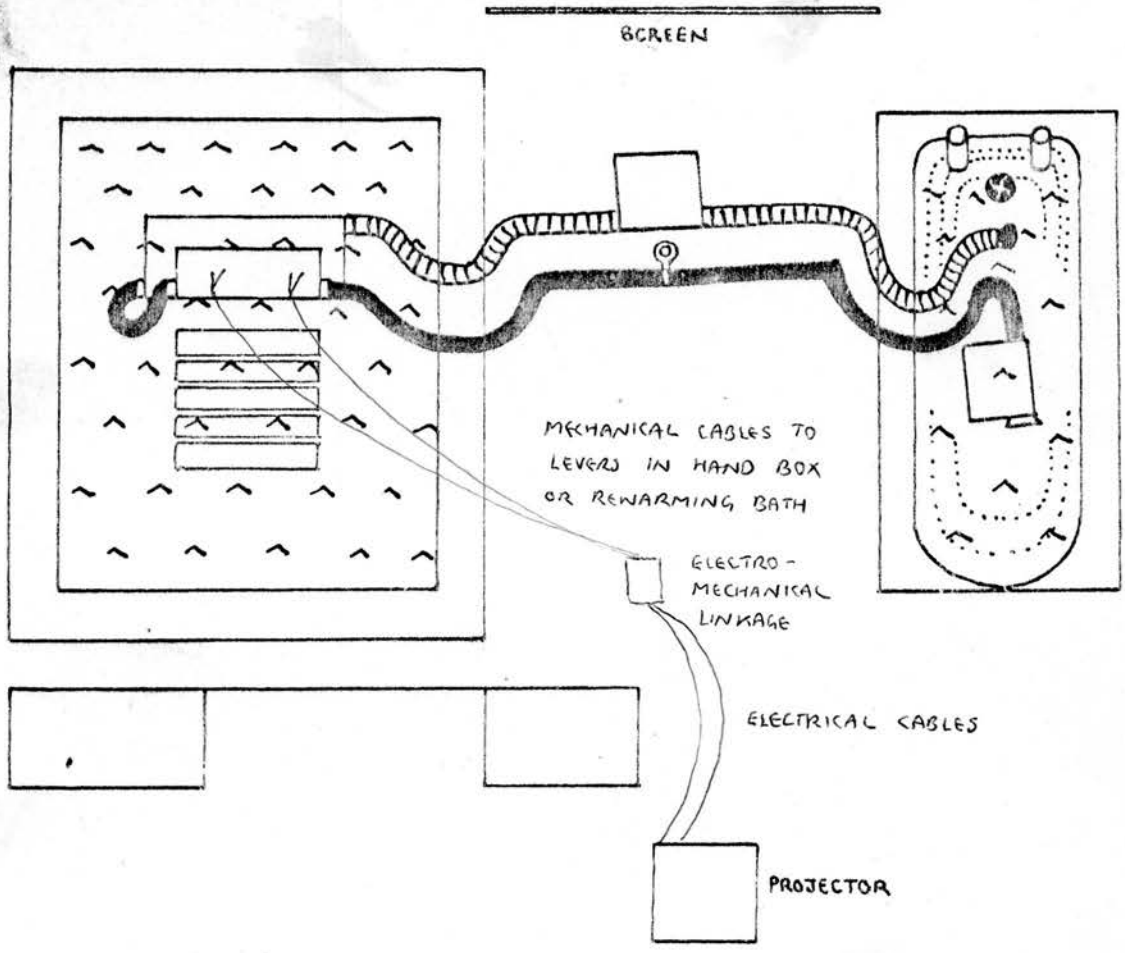


FIGURE 2: Arrangement of tank and projection apparatus.

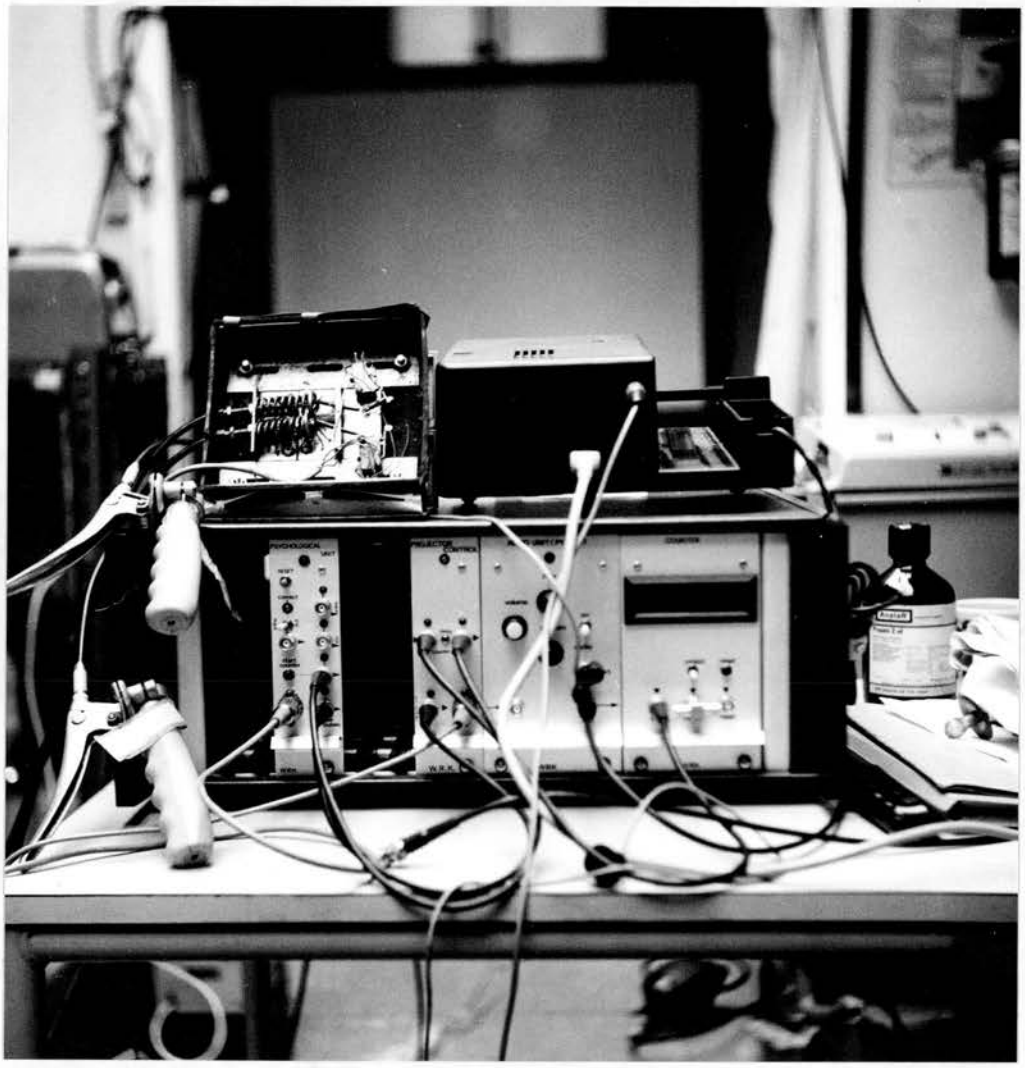


FIGURE 3: Details of projection apparatus.

order, one rack for each presentation and the subjects forewarned a minute prior to presentation.

PRESENTATION OF TESTS

Each slide was presented singly onto a screen in front of the subject (Fig. 2) and remained on view until the subject had made his response. The screen was equidistant from the immersion tank and the rewarming bath and could be easily read from both.

The timing of presentation of the six slide racks at intervals during the immersion was chosen to elicit information about the effects of peripheral stimulation by cold and by falling deep body temperature. In Figure 1, the decrease in aural temperature throughout immersion, the 'afterdrop', and on rewarming for one subject can be seen.

INFORMATION FROM DIFFERENT RACK PROJECTIONS

Rack 1

Practice - Baddeley had shown that an initial trial of the AB reasoning test was necessary, and that practice effects were negligible thereafter. The necessity of practice for the mental arithmetic tests was mainly in order to familiarise the subjects with the number, order and style of the digits.

Rack 2

Pre-immersion - The subjects were warm and dry but dressed in swimming trunks and instrumented. This test was carried out sitting in a chair facing the screen.

Rack 3

Immediately on immersion - This test might be expected to show the immediate distracting effects of cold water while deep body temperature was still normal.

Rack 4

At the end of cold immersion, while still in the tank - Subjects at this stage had a low aural temperature and their skin temperature was that of the water, i.e. either 15°C or 29°C.

Rack 5

Two to three minutes after the start of rewarming in a bath at 41°C - This test is the crucial one for demonstrating mental effects due to a low deep body temperature alone. Subjects were undergoing the post-immersion 'afterdrop' while their skin was warm and they felt comfortable.

Rack 6

At the end of rewarming while in the hot bath - By this stage the subjects had regained their pre-immersion aural temperature and felt warm and comfortable. The results of this test would be compared with the results from Rack 5 to determine the effects of a reduced brain temperature.

MEASUREMENT OF RESPONSES

The subject held a bicycle grip and lever in each hand. The levers were connected mechanically by cables to an electronic digital clock

(accurate to 0.01 s). The main reason for a mechanical linkage rather than a direct underwater electronic switch was one of electrical safety (Fig. 3).

The electronic clock, projector, linkages from the cables and controls for this apparatus were constructed into a single assembly which could be operated by one person. The clock started automatically as each slide was presented. For each calculation problem, the subject pressed the right-hand lever to record response time as soon as he reached a solution and immediately called out his answer, which was noted by the observer to be right or wrong on a standard sheet (see Appendix), and the time on the clock recorded. For AB reasoning problems, the subject pressed the right-hand lever if he thought the statement was true or the left if he thought it was false. Lights corresponding to each lever were then illuminated on a panel in front of the experimenter and the clock automatically stopped. The experimenter then recorded the subject's answer and time on a standard sheet before resetting the clock to zero and projecting the next slide. Subjects were unable to see the clock and no feedback of performance was given by the experimenter.

Subjects

Seventeen subjects were immersed for an hour in water at 15°C. Another 17 subjects were immersed for 180 minutes in water at 29°C (these were also taking part in the hand and foot cooling experiments). The tests were always carried out when the hands were at 29°C.

Controls

A separate group of the same tests was carried out on a further 6 subjects. The times of presentation were equivalent to the one-hour immersions, but the subjects were fully clothed and seated on a chair between the hot and cold baths.

Results

Tests of significance were carried out using student's t-test or related t-test where necessary.

RESULTS

EFFECTS OF REDUCED DEEP BODY TEMPERATURE ALONE (see Tables 1 and 2 and Figs. 4, 5 and 6)

These tests were performed shortly after the subject left the cold bath and had entered the warm bath at 41°C, when sensations of cold and shivering had ceased.

The time taken to complete each test is expressed as a percentage of the time needed by the subject to perform a similar test in the warm water after restoration of normal body temperature ($\pm$ 0.3°C).

There was a tendency for the subjects with lower body temperatures to take longer in completing the tests. This was seen in the relationship between body temperature and performance for the double-digit calculation after 15°C immersion ($p < 0.01$, $r = -0.72$) and when the times for the 29°C and 15°C immersion were analysed together ($p < 0.001$, $r = -0.58$). The time to perform the AB reasoning test was also significantly slower, but only when the times for 29°C and 15°C immersions were combined ($p < 0.01$, $r = -0.422$). There was no significant

TABLE 1: Means + standard errors for tests of calculation and reasoning

TEST	GROUP	ARITHMETIC 1		ARITHMETIC 2		AR	
		SCORE x SE	TIME x SE	SCORE x SE	TIME x SE	SCORE x SE	TIME x SE
Pre-immersion	Control	9 (0.4)	50 (5.8)	3.8 (0.3)	106.3 (14.6)	7 (0.3)	20.3 (2.3)
	15°C	9.4 (0.2)	56.1 (7.2)	4.2 (0.4)	134.8 (20.4)	5.9 (0.4)	44.4 (8.3)
	29°C	9.8 (0.1)	56.6 (5.7)	4.0 (0.3)	113.4 (10.7)	7.1 (0.2)	34.2 (3.8)
Immersion 3 mins	Control	9.2 (0.4)	50.4 (5.8)	4.5 (0.5)	104.8 (11.3)	7.5 (0.2)	23.7 (3.4)
	15°C	9.3 (0.3)	67.8 (8.7)	4.4 (0.4)	134.0 (19.9)	5.9 (0.4)	51.7(10.2)
	29°C	9.5 (0.2)	61.2 (6.5)	4.6 (0.3)	123.8 (16.8)	7.3 (0.2)	43.3 (6.0)
Immersed end	Control	9.7 (0.2)	41.9 (2.8)	4.5 (0.5)	101.6 (17.6)	7.8 (0.2)	19.4 (2.4)
	15°C	8.9 (0.3)	62.6 (8.0)	4.8 (0.2)	123.6 (23.6)	5.6 (0.5)	36.2 (0.2)
	29°C	9.5 (0.2)	54.8 (4.9)	4.8 (0.3)	111.3 (11.3)	6.9 (0.3)	34.1 (3.4)
Hot bath 3 mins	Control	9.7 (0.2)	42.4 (1.8)	4.5 (0.6)	87.7 (10.9)	7.8 (0.2)	21.4 (2.2)
	15°C	9.5 (0.2)	67.6(10.1)	4.7 (0.3)	122.2 (18.6)	7.0 (0.3)	55.7(12.9)
	29°C	9.6 (0.2)	56.6 (5.1)	4.8 (0.2)	91.6 (8.5)	7.4 (0.2)	39.0 (3.7)
Hot bath end	Control	9.8 (0.2)	57.4 (3.0)	4.3 (0.7)	89.5 (11.0)	8.0 (0)	20.8 (2.1)
	15°C	9.5 (0.2)	62.1 (7.1)	4.7 (0.3)	96.7 (13.5)	6.5 (0.3)	34.4 (4.9)
	29°C	9.5 (0.2)	59.2 (6.2)	5.2 (0.3)	100.5 (12.4)	7.5 (0.2)	32.1 (2.4)

Control group n = 6
15°C group n = 17
29°C group n = 17

TABLE 2: r values and significance for tests at the start of rewarming compared with aural temperature

GROUP	ARITHMETIC 1	ARITHMETIC 2	AB
15°C	$r = -0.25$ ($p > 0.3$)	$r = -0.72$ ($p < 0.01$)	$r = -0.40$ ($p < 0.2$)
29°C	$r = -0.27$ ($p > 0.3$)	$r = -0.43$ ($p > 0.05$)	$r = -0.42$ ($p > 0.05$)
15°C & 29°C	$r = -0.31$ ($p > 0.3$)	$r = -0.58$ ($p < 0.001$)	$r = -0.46$ ($p < 0.01$)

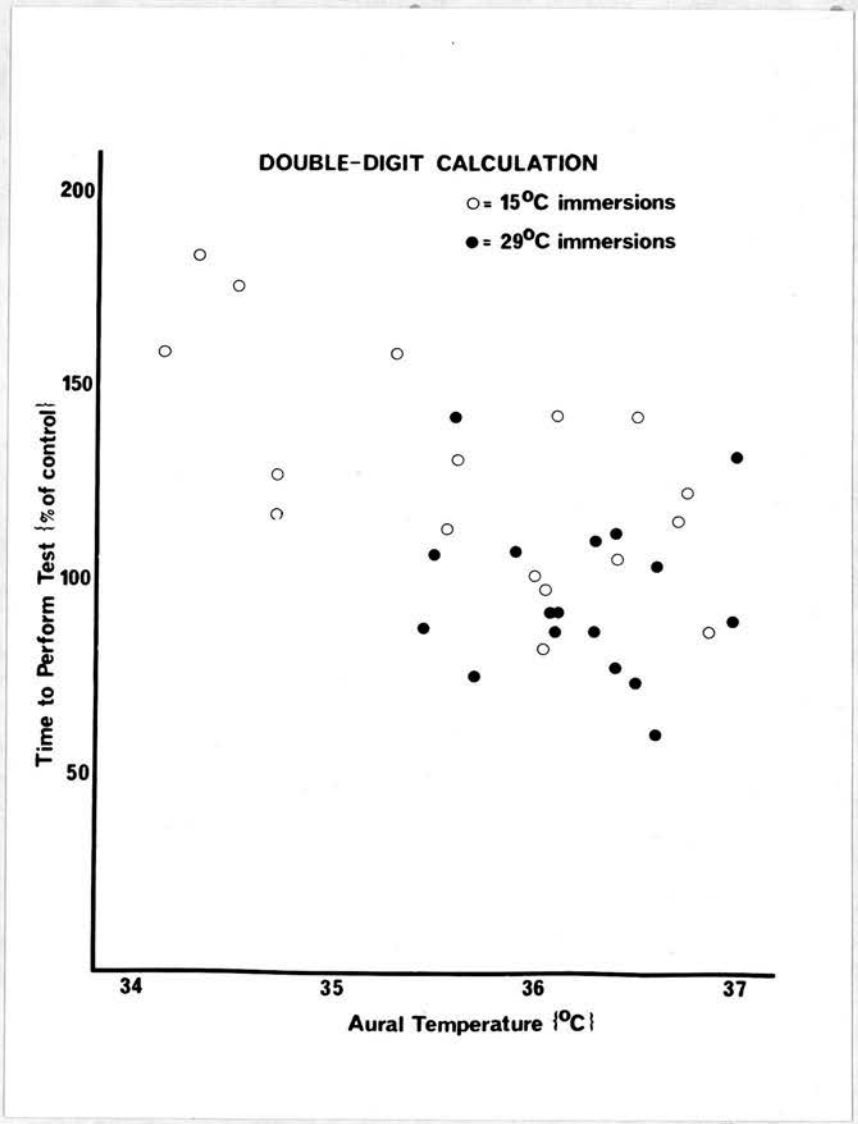


FIGURE 4: Time needed to perform double-digit calculation test in relation to core temperature with warm skin, at the start of rewarming. Time is % of time taken by same subject in a subsequent control test (mean for controls, 98.0 s) at normal body temperature.

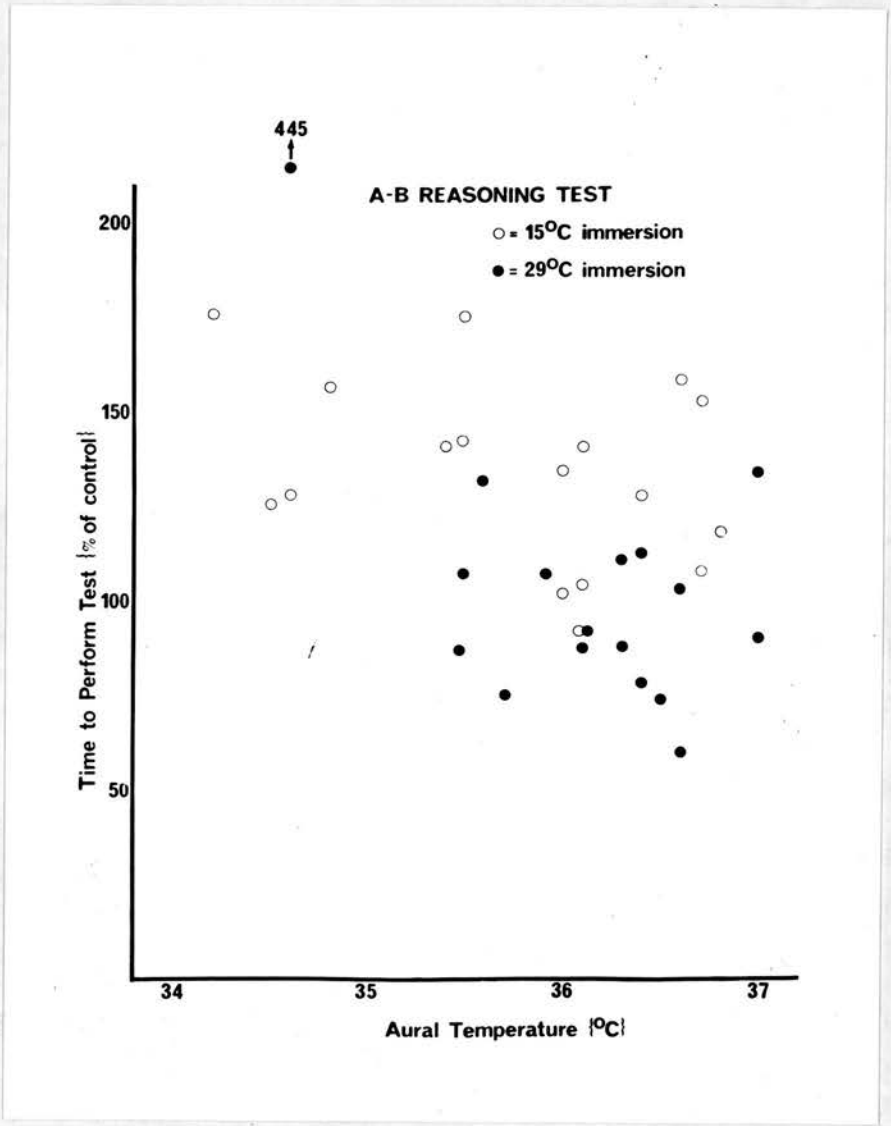


FIGURE 5: Time needed to perform AB reasoning test in relation to core temperature with warm skin, at the start of rewarming. Time is % of time taken by same subject in subsequent control test (mean for controls, 34.5 s) at normal body temperature.

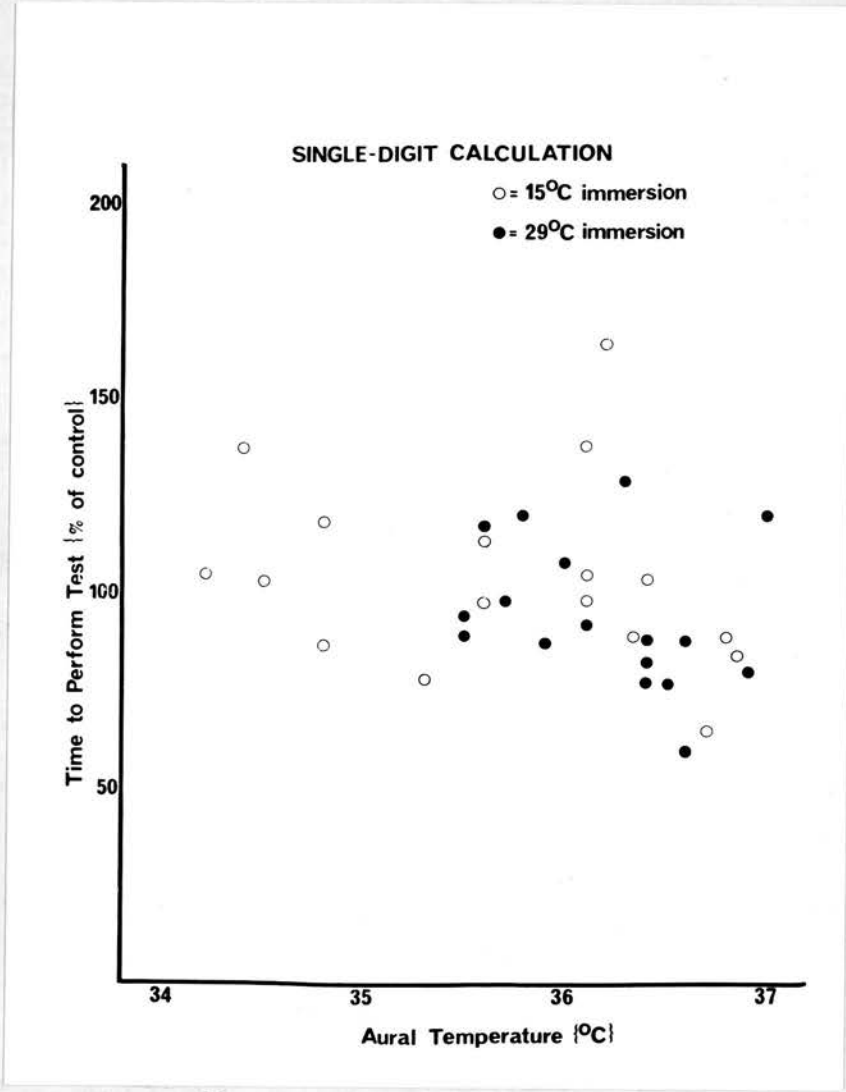


FIGURE 6: Time needed to perform single-digit calculation test in relation to core temperature, with warm skin at the start of rewarming. Time is % of time taken by same subject in subsequent control test (mean for controls 62.1 s) at normal body temperature.

relationship between time to perform tests and body temperature during the single-digit arithmetic calculation. There was no significant relationship between body temperature and time to complete the tests after restoration of normal body temperature. The number of correct answers was unrelated to body temperature.

Controls

There was no difference in scores or times for the two tests.

THE EFFECT OF A COLD SKIN AND NORMAL DEEP BODY TEMPERATURE

The tests immediately prior to, and immediately after, the start of cold immersion were compared (see Table 2).

29°C

The time taken to perform the single-digit arithmetic test in the water was significantly slower (means 56.6 $\pm$ 5.7 s and 61.2 $\pm$ 6.5 s, $p < 0.05$).

The time taken to perform the AB reasoning test was also significantly slower (means 34.2 $\pm$ 3.8 s and 43.3 $\pm$ 6.0 s, $p < 0.02$), but the control group in air were also slower on this test (20.3 $\pm$ 2.3 s and 23.7 $\pm$ 3.4 s, $p < 0.05$). The double-digit arithmetic test showed no significant differences (means 106.3 $\pm$ 14.6 s and 104.8 $\pm$ 11.3 s).

15°C

The single-digit arithmetic test was significantly slower (56.1 $\pm$ 7.2 s and 67.8 $\pm$ 8.7 s, $p < 0.05$).

The AB reasoning test and double-digit tests showed no significant differences.

Scores

There were no differences in scores for any of the groups.

Controls

There were no differences for scores or times.

THE EFFECT OF A COLD SKIN AND A LOW DEEP BODY TEMPERATURE

The tests performed at the end of immersion immediately prior to leaving the cold tank, and those shortly after entering the warm bath, were compared.

29°C

The AB reasoning test was significantly faster in the cold tank (34.1 ± 3.4 s and 39.0 ± 3.7 s, $p < 0.05$).

The double-digit arithmetic test was significantly slower in the cold tank (111.3 ± 11.3 s and 91.6 ± 8.5 s, $p < 0.005$).

Single-digit addition showed no change.

Mean aural temperature was 36.3 ± 0.15°C in the 29°C tank and 36.2 ± 0.16°C in the 41°C bath.

15°C

There were no significant differences in the times for any of the groups (mean aural temperatures 36.25 ± 0.24°C in 15°C and 35.65 ± 0.2°C in 41°C).

Scores

There were no significant differences in scores for any of the groups.

Controls

There were no differences in the scores or times.

CONTROLS FOR LEARNING

A comparison of the times for the pre-immersion tests and the last tests at the end of the experiment showed that the control group was faster at the end of the experiment for double-digit addition (106.3 ± 14.6 s and 89.5 ± 11 s, $p < 0.05$) but not for the other two tests. The group immersed at 15°C were faster for the double-digit test (134.8 ± 20.4 s and 96.7 ± 13.5 s, $p < 0.05$) and slower for the single-digit tests (56.1 ± 7.2 s and 62.1 ± 7.1 s, $p < 0.05$), but the same for AB reasoning.

The group immersed in 29°C water did not show any differences for these two sets of tests. Scores were not different for any group.

Although there may have been a tendency to increase in speed with practice for the double-digit addition, there was no difference in times between the last two tests, indicating that learning effects had been eliminated by the time these tests were performed.

DISCUSSION

These findings show that hypothermia can cause slowing of mental processes, such as mental arithmetic addition and simple reasoning

problems. Accuracy is not affected provided sufficient time is allowed to complete the task.

Slowing is progressive below deep body temperatures of around 36°C and is substantial. For example, the time required to add four double digits together at 34°C is almost twice as long (175%) as that for normal body temperature.

This magnitude of slowing of complex tasks compares with the 120% slowing found by Davis, Baddeley and Hancock (1975) on similar arithmetic problems performed in cold water, the 150% slowing on coding problems and 300% on psychomotor tests found by Biersner (1976) and the 130% and 155% slowing described by Bowen (1968) on two series of cold exposures.

However, the difficulties in analysis of previous authors' data due to inadequate deep body temperature measurement and presentation of temperature data as a group mean, when all divers may not have been hypothermic, have been previously described in Chapter Four.

The design of the present experiments leaves no doubt that mental slowing is due to a low brain temperature and the degree of slowing is proportional to the lowered temperature. A distraction effect of cold water on the skin has been the favoured explanation for mental slowing described by these other authors. A similar slowing was found in the present experiments but the slowing was much less, of the order of 10%, and only demonstrable for the first test immediately after entering the cold water. No slowing was seen for the second and third tests of the same rack, performed a few minutes after the first tests. This 'distraction' effect was present in the first tests for both the 29°C and 15°C immersions, suggesting that the novel situation of being in the tank, rather than the water temperature, was responsible.

Again, it seems unlikely that distraction by cold has other than a brief and temporary effect because there was no difference in results between the tests in the cold bath at the end of the immersion and those a few minutes later in the warm bath. Aural temperatures were 36.25 $\pm$ 0.24°C in the cold bath at that stage and 35.65 $\pm$ 0.22°C after 3 minutes in the hot bath (15°C immersion).

The 20% slower speed of the double-digit test at the end of the 29°C immersion compared with those in the hot bath, with aural temperatures of 36.32 $\pm$ 0.15°C and 36.19 $\pm$ 0.16°C respectively, may have been due to boredom or fatigue, since by that stage subjects had been immersed for 180 minutes rather than the 60 minutes of the 15°C immersions.

The strength of forearm muscular contraction diminishes progressively below muscle temperatures of 27°C (Clarke, Hellon and Lind, 1958) and is the temperature of forearm muscle after 30 minutes' immersion in 18°C water described in the same paper. A reduction in manual dexterity will have been part of the explanation for reduced response times of divers cooled in 16.7°C water (Bowen, 1968) and probably of those cooled in 21°C water (Stang and Weiner, 1970) and 25.6°C water (Davis, Baddeley and Hancock, 1975).

The forearms and hands of the subjects in the present experiments were maintained in water at 29°C by means of a separate water circulation in a hand box, and impairment of muscular contraction by cold cannot therefore account for the observed performance decrement in the cold. In addition, muscular activity was limited to pulling a fist-sized lever, and no fine movements or complicated manoeuvres were designed into the experiment. Previous investigators have without exception attempted authenticity of diving conditions by using breathing apparatus and insulating suits.

The disadvantages inherent in thus attempting authenticity are not only that divers were to some extent protected from cold, and may not have been hypothermic despite having a cold skin, but also that carbon dioxide build-up in the breathing apparatus, although unlikely, could not be excluded.

Subjects in the experiments described here cooled to levels adequate to show mental effects because they wore no thermal protection, and build-up of carbon dioxide was excluded because they breathed room air.

In summary, these experiments clearly demonstrate a dramatic impairment of mental abilities with even modest hypothermia, where deep body temperature was not allowed to fall below 34°C. The impairment is manifested as a slowing of the abilities to perform simple arithmetical and reasoning tests, rather than inaccuracy, and approaches twice as long as the time necessary for the same tests at normal body temperatures.

The experimental design enabled the described mental slowing to be attributed directly to brain cooling rather than a distraction effect of a cold skin, impaired manual dexterity by cold, or to carbon dioxide retention.

REFERENCES

BADDELEY AD (1968). A 3-minute reasoning test based on grammatical transformation. Psychon Sci 10, 341-342.

BIERSNER RJ (1976). Motor and cognitive effects of cold water immersion under hyperbaric conditions. Hum. Factors 18, 229-304.

BOWEN HM (1968). Diver performance and the effects of cold. Hum. Factors 10, 445-464.

CLARKE RSJ, HELLON RF, LIND AR (1958). The duration of sustained contractions of the human forearm at different muscle temperatures. J Physiol 143, 454-473.

DAVIS FM, BADDELEY AD, HANCOCK TR (1975). Diver performance: the effect of cold. Undersea Biomed Res 2, 195-213.

STANG PR, WEINER EL (1970). Diver performance in cold water. Hum. Factors 12, 391-399.

CONCLUSIONS

1. During immersion of fit, young adults for three hours in lukewarm water at 29°C, there was little shivering or discomfort, but deep body temperature progressively fell, with a range of 0.5°C to 1.4°C fall in the subjects examined. There was no evidence of temperature stabilisation during this time. The rate of temperature drop was inversely related to the mean subcutaneous fat thickness.

   All subjects were capable of preventing such a fall in deep body temperature but did not do so until a stimulus of colder water at 12°C was applied to the hands and feet. In two further subjects, a stimulus of water at 20°C had a similar effect, while one of 25°C had a less marked effect. When the hands and feet were cooled, the rest of the body remaining in lukewarm water, deep body temperature ceased to fall and often rose.

   An increase in metabolic rate and heat production by shivering was the explanation for this halting of 'insidious' loss of deep body temperature, and there was little change in heat loss through the skin. Cold-acclimatised subjects tended to shiver less and reduce forearm heat loss, but heat loss through other regions remained similar to that seen in lukewarm water prior to hand and foot cooling.

   The reason for 'insidious' hypothermia during immersion in 29°C water is likely to be inadequate stimulation of skin thermoreceptors by such mild cooling. Despite major falls in deep body temperature, little or no metabolic response was induced. When the uniform pattern of 29°C skin temperature was disrupted by local hand and foot chilling, the input of extra information from these

small areas, totalling 12% of the body surface, was enough to provoke an increase in metabolic rate sufficient to raise deep body temperature.

2. Below deep body temperature of 36°C or less, there was a progressive slowing of the ability to perform simple reasoning and arithmetical problems. When deep body temperature approximated 34°C, the time taken to perform these tests nearly doubled that taken at normal body temperature. Accuracy of addition or simple reasoning was not affected in this temperature range, provided enough time was given to complete the problems.

Similarly, the ability to form new memories, as measured by scores achieved on a standard memory passage, was seriously affected when deep body temperature fell below about 36°C, and progressively worsened with lower temperatures. The ability to recall previously learned information at such temperatures was unimpaired.

These cognitive studies are of interest because they clearly show that cold initially affects only specific kinds of cerebral activity. Besides the theoretical questions of central processing raised by this work, very practical advice can also be given to divers or others exposed to cold. It is imperative that deep body temperature is kept above 36°C, for below this temperature there is a measurable decline in the ability to think quickly or to remember new information.

Immersion in lukewarm water, or diving suit heating with water of this temperature, is therefore doubly dangerous. Not only is there a progressive development of hypothermia, of which a person is

unaware, but there is also a serious decline in mental abilities which occurs early in the development of such hypothermia.

APPENDIX ONE

—ooOoo—

CALIBRATION OF HEAT FLOW BANDS

(Gin AR, Hayward MG, Keatinge WR (1980). Method for measuring regional heat losses in man. J. Appl. Physiol. 49, 533-535)

The electrical output of the copper-constantan thermocouple heat flow band is not proportional to the heat loss that takes place through adjacent skin not covered by the device, because of the insulation provided by the band itself.

There is, however, a linear relation between the reciprocal of the output of the device and the reciprocal of heat loss from adjacent skin not covered by the device, for any given temperature difference between body core and water outside the body.

$$\frac{T}{H} = \frac{T}{KE} - I_D$$

T = temperature gradient from core to water

H = heat flow per unit area

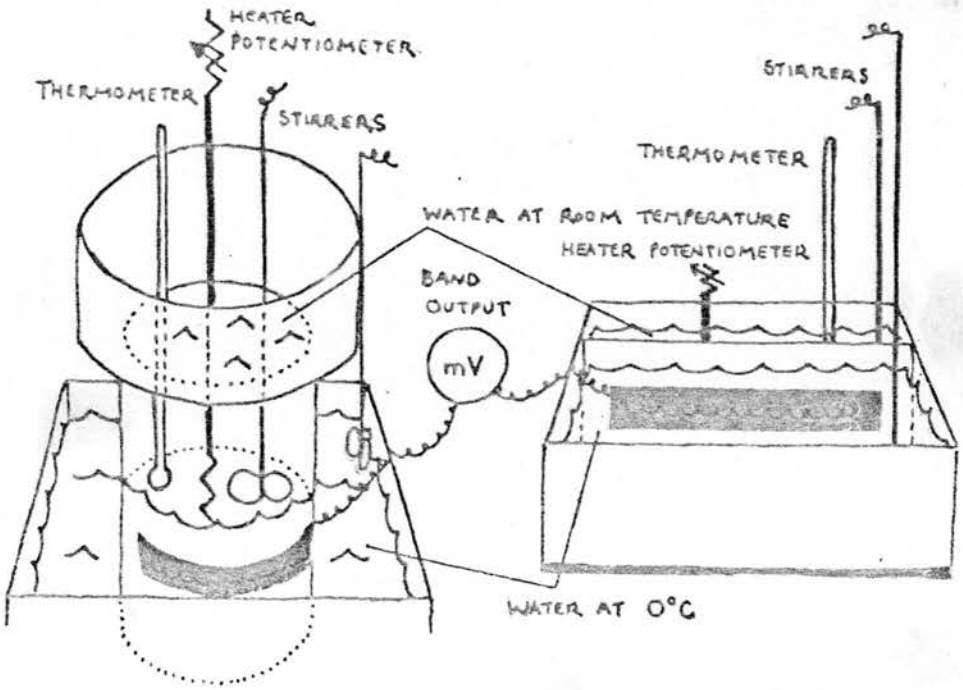
K = constant

E = electromotive force generated by the band

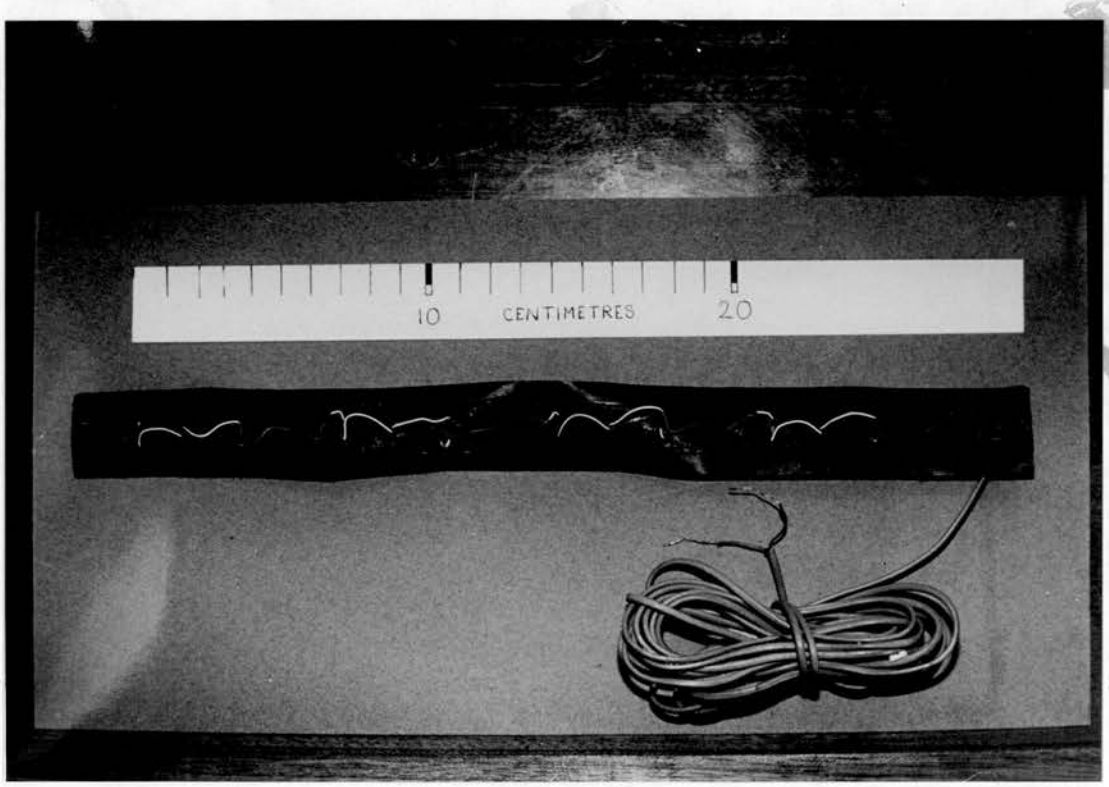
I_D = insulation due to band itself

A graph of $\frac{T.°C.m^2.W^{-1}}{H}$ vs $\frac{T.°C.\mu v^{-1}}{E}$ was constructed for each heat flow band utilising four measurements. The heat flow band was applied to the exterior of a perspex cylinder and immersed in stirred water kept at 0°C by crushed ice. The interior of the cylinder was filled with water at room temperature, and the output of a heater placed within the cylinder adjusted using a potentiometer until the voltage required to keep the water at room temperature remained constant. Measurements of T/H and T/E provided one point on the graph. Two further points were obtained using perspex cylinders of different thickness. A final (zero) point was obtained by using the band to seal a rectangular opening between two stirred water baths at different temperatures, and measuring directly T as the difference in temperature between the water baths and

voltage output of the band when there was no insulation in series with it.



2. Calibration box and cylinder(s).



1. part of continuous type of heat flow band.

READINGS

1. THIN CYLINDER S.A. ..... m^2 CORRECTED S.A. ..2352.. m^2

T_{o_C} CYL	T_{o_C} TANK	$\Delta T\ ^{o}C$	μv	$\frac{\Delta T}{\mu v}$	SOURCE VOLTS	SOURCE CURRENT		WATTS	KCALS. MIN^{-1}	CORRECTED KCALS. MIN^{-1}	$H=KCALS.\ MIN^{-1}.M^{-2}$	$\frac{\Delta T}{H}$
						$\frac{mv}{mohms}$	iamps					
20.4	0.00	20.4	718	.028	135	7.87/7.5	2.005	141.66	2.031	2.051	8.720	2.34

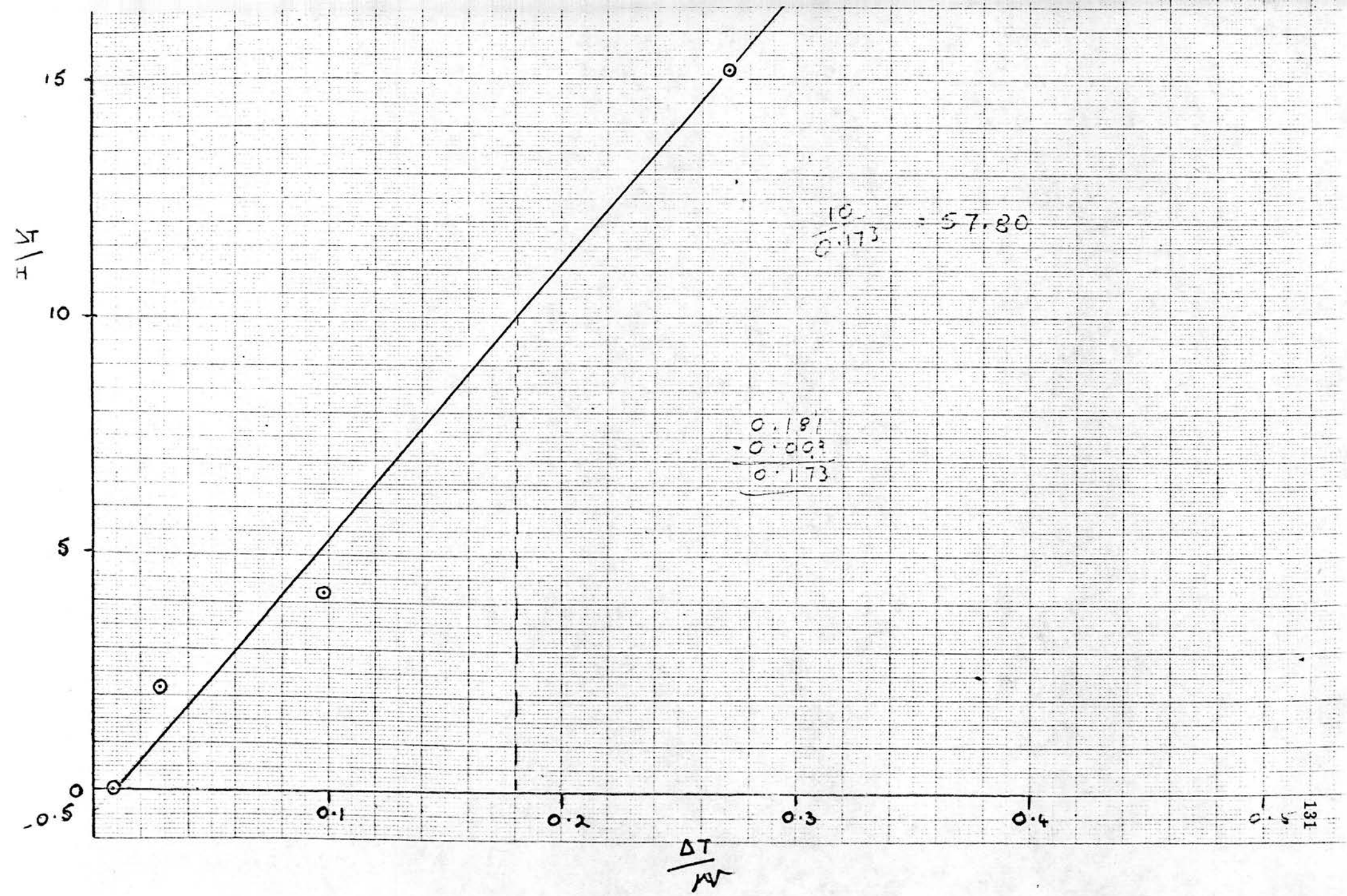
2. MIDDLE CYLINDER S.A. ..... m^2 CORRECTED S.A. ..2328.. m^2

T_{o_C} CYL	T_{o_C} TANK	$\Delta T\ ^{o}C$	μv	$\frac{\Delta T}{\mu v}$	SOURCE VOLTS	SOURCE CURRENT		WATTS	KCALS. MIN^{-1}	CORRECTED KCALS. MIN^{-1}	$H=KCALS.\ MIN^{-1}.M^{-2}$	$\frac{\Delta T}{H}$
						$\frac{mv}{mohms}$	iamps					
20.8	0.6	20.2	206	0.098	97	5.93/7.5	0.791	76.73	1.099	1.119	4.807	4.20

3. THICK CYLINDER S.A. ..... m^2 CORRECTED S.A. ..2967 m^2

T_{o_C} CYL	T_{o_C} TANK	$\Delta T\ ^{o}C$	μv	$\frac{\Delta T}{\mu v}$	SOURCE VOLTS	SOURCE CURRENT		WATTS	KCALS MIN^{-1}	CORRECTED KCALS. MIN^{-1}	$H=KCALS\ MIN^{-1}.M^{-2}$	$\frac{\Delta T}{H}$
						$\frac{mv}{mohms}$	iamps					
17.3	0.5	16.8	62	0.271	50	3.22/7.5	0.429	21.47	0.308	0.328	1.105	~~0.05~~
												15.19

4. No INSULATION $\Delta T\ ^{o}C$ 9.65 μv 1090 $\frac{\Delta T}{\mu v}$ 0.0089 MISC. 18


15
10
5
0
-0.5
H/ΔT
0.1
0.2
0.3
0.4
ΔT/μV
10/0.173 = 57.80
0.181
-0.008
0.173

3. Example of calibration of one device.

APPENDIX TWO

—ooOoo—

THE ZERO-GRADIENT AURAL THERMOMETER

(Keatinge WR, Sloan REG (1975). Deep body temperature from aural canal with servocontrolled heating to outer ear. J. Appl. Physiol. 38, 919-921)

The zero-gradient aural thermometer is a method of measuring deep body temperature. Sublingual temperature is unreliable, rectal probes demonstrate a lag behind changes in deep body temperature, and oesophageal probes are uncomfortable. A simple thermistor placed in the aural canal is seriously affected by cooling caused by temperature gradients along the aural canal.

The zero-gradient thermometer consists of a pair of matched thermistors whose outputs are compared by a differential amplifier. One thermistor is placed deep within the aural canal and the other in contact with the ear skin. Gradients between the two are abolished by a heating pad on the ear, the current of which is controlled by the differential amplifier.

The zero-gradient aural thermometer.

APPENDIX THREE

—ooOoo—

HAND AND FOOT COOLING AT 25°C AND 20°C WHILE IMMERSED IN 29°C WATER

In Chapter Three, experiments are described where the progressive decline in deep body temperature seen during 29°C immersions was reversed by further cooling of the hands and feet only in 12°C water. While chilling the hands and feet at this temperature was undoubtedly effective in increasing metabolic rate by shivering, it was also very uncomfortable for the subjects.

If hand and foot cooling was to have any practical implications, the degree of cooling would have to remain tolerable. In addition, it was of theoretical interest to see the amount by which the hands and feet could be cooled before alterations in response were evident.

Two subsequent immersions were therefore undertaken, each with a subject acting as his own control, cooling in 29°C water and undergoing hand and foot cooling to 25°C and 20°C.

Because there were two periods of peripheral chilling rather than a single one at 12°C, the immersions were longer, at 300 minutes.

SUBJECTS

Both subjects were non-divers and had not experienced recent exposures to cold apart from immersions as control subjects in 29°C water three months previously.

SUBJECT	AGE	HEIGHT (m)	WEIGHT (kg)	MEAN SUBCUTANEOUS FAT THICKNESS
SC	22	1.63	66.3	10.5
NVS	29	1.83	77	7.5

METHODS

Subjects were medically examined and mean subcutaneous fat thickness determined ultrasonically (Wells-Krautkramer model VSM 2F) from measurements at four sites (Chapter Two).

Instrumentation consisted of electrocardiogram leads, heat flow bands on hands and feet, forearms, thigh, upper arm and trunk, and the zero-gradient aural thermometer.

For the duration of the immersion, they were seated in the immersion tank containing water thermostatically controlled to 29°C $\pm$ 0.1°C.

Hands and feet were placed in perspex boxes with soft interseals at wrists and ankles. The temperature of the water circulating in the boxes was controlled by adding hot or cold water to a remote reservoir.

The reference thermocouple was within the tank and box temperatures and heat flow band outputs measured on a high resistance (500 MΩ) digital voltmeter (Fenlow Electronics). Metabolic rates were calculated from the measurement of expired oxygen collected in Douglas bags and analysed with a paramagnetic oxygen analyser (Beckman).

When heat flow from the hands and feet had stabilised at 90 minutes after the start of immersion, water at 25°C was circulated around the hands and feet for 60 minutes. The 29°C water was then circulated around the hands and feet again for 60 minutes. Following this, water at 20°C was introduced into the boxes for a further 60 minutes before returning the box temperature to 29°C.

RESULTS

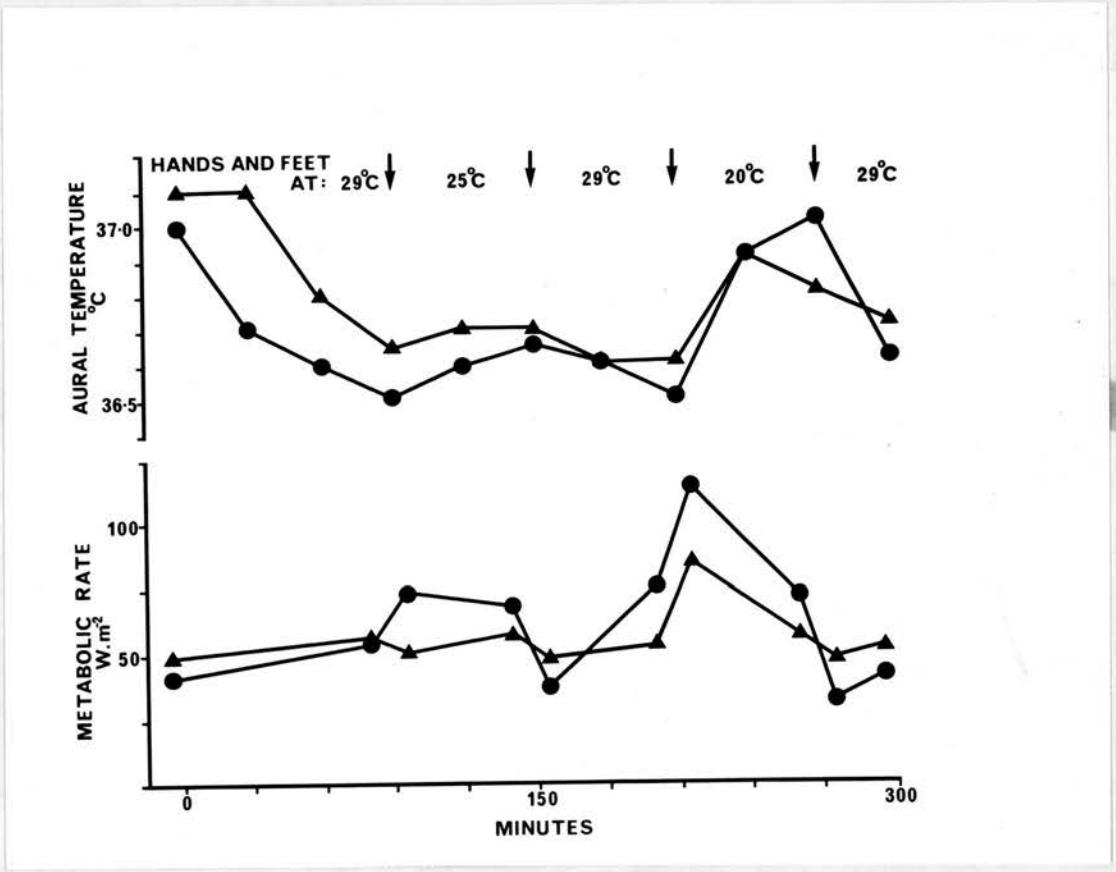
Aural temperature

During the first 90 minutes of immersion, aural temperature fell in both subjects by approximately 0.4°C per hour, with no evidence of stabilisation of deep body temperature. On the introduction of 25°C water into the boxes, the aural temperature rose from 36.5°C to 36.6°C in the male, and from 36.65°C to 36.7°C in the female. On cessation of 25°C circulation and re-introduction of a uniform skin temperature of 29°C, the temperature of both resumed the previous decline. When 20°C water was then circulated, their aural temperatures rose from 36.5°C to 37°C in the male, and from 36.6°C to 36.9°C in the female. The temperature rise was sustained in the male during the hour of cooling at 20°C but declined somewhat to 36.8°C in the female.

Finally, with the return to a uniform skin temperature of 29°C, the aural temperature of both declined again to that seen before 20°C hand and foot cooling.

Metabolic rates

The metabolic rates of both subjects changed little during the first 90 minutes, that of the male being approximately 50 $W.m^{-2}$ and the female 40 $W.m^{-2}$. The metabolic rate of the man rose to 69 $W.m^{-2}$ with 25°C hand and foot cooling, and to 110.0 m^{-2} with 20°C cooling, with a return to 50 $W.m^{-2}$ or less in uniform 29°C skin cooling. The metabolic rate of the female subject changed little with 25°C box temperatures, but rose to 80 $W.m^{-2}$ with 20°C box temperatures, before returning to the previous rate of 40 $W.m^{-2}$ on resumption of uniform 29°C skin cooling.



Effect of cooling hands and feet in 25°C and 20°C water, during general immersion in water at 29°C, on deep body temperature and metabolic rate. Two subjects, both unacclimatised to cold: ●, male subject; ▲, female subject.

Interpretation

The rise in aural temperature with 25°C and 20°C hand and foot cooling was due to an increased metabolic rate by shivering. The increases seen with temperatures only 4°C below those of the trunk and upper limbs were minimal, but the increases seen at 20°C were of the same order as those with much more severe chilling at 12°C (Chapter Three).

This indicates that severe chilling of the hands and feet is not necessary to stimulate the return of an adequate thermoregulatory response after 'insidious' cooling in tepid water. The possible reasons for this are discussed in Chapter Three.

APPENDIX FOUR

DATA RECORDING SHEETS

MEDICAL

NAME: *AGE:* *M/F:*

OCCUPATION:

PAST HISTORY OR FAMILY HISTORY OF:

Frostbite Chilblains

Cold injury Raynauds

Local reactions to cold Collapse on cold exposure

Heart disease Chest infection/disease

Ear infection Any other illness or operation

Date and result of last chest x-ray

ANY CURRENT SYMPTOMS:

Cough Chest pain

Sputum Ankle swelling

Shortness of breath Ear discharge/deafness

Weight loss Vomiting/diarrhoea

ANY CURRENT MEDICATION (including aspirin):

Examination

Anaemia Cyanosis Clubbing JVP

Ankle oedema AB HS Pulse BP

Chest expansion Percussion Auscultation

NAME: ..... AGE: ..... M/F: .....

HEIGHT /m: Standing .....

Sitting .....

WEIGHT /kg: .....

INDIRECT S.A. /m^2: .....

SKINFOLD THICKNESS:

Biceps					
Subscap.					
Abdomen					
Subcostal					

MEAN SKINFOLD THICKNESS: .....

BAND LENGTHS /cm:

Hand .....

Foot .....

Forearm .....

Opp. forearm .....

Calf .....

Mid trunk .....

MISC.31

NAME: ..... *DATE:* .....

TIME AFTER IMMERSION /min	VOLUME OF GAS EXPIRED /l	CORRECTED VOLUME /l	TIME OF COLLECTION /min	$\dot{V}_E$ /l.min^{-1}	$\dot{V}_E$ (STP) /l.min^{-1}	D	% O_2	$\dot{V}_{O_2}$ /l.min^{-1}	METABOLIC RATE /Watts

MISC.30

NAME: ..... DATE: .....

HEAT FLOW BANDS

TIME AFTER IMMERSION /min	$^{-}\Delta T$ /°C	FOREARM TEMP /μV	H /W.m^{-2}	OPPOSITE FOREARM TEMP /μV	H /W.m^{-2}	CALF TEMP /μV	H /W.m^{-2}	MID-TRUNK TEMP /μV	H /W.m^{-2}

MISC. 35

NAME: ..... DATE: .....

THERMOCOUPLES - TEMPERATURE

TIME AFTER IMMERSION /min	WATER TEMP IN TANK /°C	Δ HAND BOX TEMP /°C	ACTUAL HAND BOX TEMP /°C	Δ FOOT BOX TEMP /°C	ACTUAL FOOT BOX TEMP /°C	Δ HAND SKIN TEMP /°C	ACTUAL HAND SKIN TEMP /°C	Δ FOOT SKIN TEMP /°C	ACTUAL FOOT SKIN TEMP /°C

MISC.33

MEMORY PASSAGE - SCORE

NAME: .....

CONTROL/EXPERIMENT

TYPE	*NAME*	*DEPTH*	*FLOOR*	*DANGER*
Liner	Olympus	24 M	Gulley	Currents
Yacht	Nipper	11 M	Sand	Low visibility
Cabin cruiser	Limpet	5 M	Cliff foot	Swell

OMISSIONS: .....

TRANSFERS: .....

FABRICATIONS: .....

INACCURACIES: .....

CONTROL/EXPERIMENT

Fishing boat	Lady Lucy	3 M	Kelp	Poison fish
Battleship	HMS Intrepid	20 M	Shingle	Sharks
Coaster	Mermaid	50 M	Rocks	Sharp metal

OMISSIONS: .....

TRANSFERS: .....

FABRICATIONS: .....

INACCURACIES: .....

ARITHMETIC TEST I (single digit strings)

SUBJECT'S NAME: .....

DATE: .....

TEST SERIES: PRACTICE

SUBJECT'S ANSWER	*CORRECT ANSWER*	*SCORE*	*TIME*
1.	26	✓	6.28
2.	31	–	4.32
3.	26	✓	4.67
4.	24	✓	5.74
5.	22	✓	3.68
6.	31	✓	6.39
7.	18	✓	4.28
8.	24	✓	3.93
9.	25	—	4.11
10.	21	✓	3.57
	TOTAL	____	____

T^{Ear} T^{R}

2.5 - 3 min.

ARITHMETIC I

SUBJECT'S NAME: .....

DATE: .....

TEST SERIES: PRE-IMMERSION

SUBJECTS ANSWER	*CORRECT ANSWER*	*SCORE*	*TIME*
1.	23	23	5.04
2.	23	21	4.99
3.	18	✓	3.04
4.	34	✓	5.25
5.	23	✓	3.05
6.	31	✓	3.35
7.	32	✓	3.50
8.	31	✓	5.57
9.	25	—	2.93
10.	27	✓	3.78
	TOTAL		

T^{Ear} T^{R}

MISC.37/2

1½

46°

14.5°

7708

9874.

7708

2166 μV / 315°

6876

69 μV / °C.

ARITHMETIC I

SUBJECT'S NAME: .....

DATE: .....

TEST SERIES: IMMEDIATELY ON IMMERSION (3 min)

SUBJECT'S ANSWER	*CORRECT ANSWER*	*SCORE*	*TIME*
1.	27		
2.	27		
3.	35		
4.	28		
5.	27		
6.	30		
7.	16		
8.	24		
9.	24		
10.	22		
	TOTAL	____	____
		____	____

T^{Ear} T^{R}

MISC.37/3

ARITHMETIC I

SUBJECT'S NAME: .....

DATE: .....

TEST SERIES: IMMERSED - END OF RUN *TIME:* .....

	SUBJECT'S ANSWER	*CORRECT ANSWER*	*SCORE*	*TIME*
1.	23	23		4·76
2.	28	28		4·16
3.	24	24		5·52
4.	25	25		3·33
5.	16	16		4·05
6.	31	31		4·69
7.	21	21		6·26
8.	22	22		4·19
9.	22	22		2·86
10.	20	20		3·53
		TOTAL	____	____
			____	____

T^{Ear} T^{R}

ARITHMETIC I

SUBJECT'S NAME: .....

DATE: .....

TEST SERIES: REWARMING, 3 min from start

SUBJECT'S ANSWER	*CORRECT ANSWER*	*SCORE*	*TIME*
1.	29		
2.	24		
3.	27		
4.	22		
5.	33		
6.	21		
7.	22		
8.	18		
9.	24		
10.	18	____	____
	TOTAL	____	____

T^{Ear} T^{R}

MISC.37/5

ARITHMETIC I

SUBJECT'S NAME: .....

DATE: .....

TEST SERIES: REWARMING FINAL

	SUBJECT'S ANSWER	*CORRECT ANSWER*	*SCORE*	*TIME*
1.	41	41	-	5.99
2.	20	20		3.61
3.	27	27		3.29
4.	22	22		2.61
5.	29	29		5.37
6.	25	25		4.23
7.	26	26		4.32
8.	30	30		11.76
9.	30	30		7.41
10.	32	32		4.97
		TOTAL	____	____
			____	____

T^{Ear} T^{R}

MISC.37/6

ARITHMETIC TEST II (2 digit strings)

SUBJECT'S NAME: .....

DATE: .....

TYPE OF IMMERSION: PRACTICE

SUBJECT'S ANSWER	*CORRECT ANSWER*	*SCORE (1 point per item)*	*TIME*
1.	229	249	22.38
2.	232	232	11.21
3.	195	203	12.43
4.	254	264	18.11
5.	149	149	6.79
6.	222.	321	14.79
	TOTAL	2	

T^{Ear} T^{R}

2 2½ min.

ARITHMETIC II (2 digit)

SUBJECT'S NAME: .....

DATE: .....

PRE-IMMERSION

SUBJECT'S ANSWER	*CORRECT ANSWER*	*SCORE*	*TIME*
1.	228	✓	18.98
2.	218	–	8.20
3.	319	–	7.73
4.	245	–	15.48
5.	166	✓	15.91
6.	254	–	15.84
	TOTAL		

T^{Ear} T^{R}

~ 1½

MISC.38/2

ARITHMETIC II (2 digit)

SUBJECT'S NAME: .....

DATE: .....

IMMEDIATELY ON IMMERSION (3 min)

SUBJECT'S ANSWER	*CORRECT ANSWER*	*SCORE*	*TIME*
1.	215		
2.	322		
3.	230		
4.	242		
5.	262		
6.	243		
	TOTAL	----	----

T^{Ear} T^{R}

ARITHMETIC II (2 digit)

SUBJECT'S NAME: .....

DATE: .....

IMMERSED-END OF RUN *TIME* ..... min 12·44

	SUBJECT'S ANSWER	*CORRECT ANSWER*	*SCORE*	*TIME*
1.	201	201		7·77
2.	278	278		13·42
3.	243	243		13·88
4.	209	209		8·72
5.	273	273		16·22
6.	255	255		16·10
		TOTAL	----	----

T^{Ear} T^{R}

MISC.38/4

ARITHMETIC II (2 digit)

SUBJECT'S NAME: .....

DATE: .....

REWARMING (3 min from start)

SUBJECT'S ANSWER	*CORRECT ANSWER*	*SCORE*	*TIME*
1.	173		
2.	231		
3.	151		
4.	151		
5.	184		
6.	254		
	TOTAL		

T^{Ear} T^{R}

ARITHMETIC II (2 digit)

SUBJECT'S NAME: .....

DATE: .....

REWARMING, FINAL

	SUBJECT'S ANSWER	*CORRECT ANSWER*	*SCORE*	*TIME*
1.	202	202		11·41
2.	200	200		9·79
3.	295	295		10·24
4.	203	203		
5.	252	253		13·94
6.	210	210		11·35
		TOTAL	———	———

T^{Ear} T^{R}

MISC.38/6

AB REASONING TEST

SUBJECT'S NAME: .....

DATE: .....

TYPE OF IMMERSION: Practice

	ITEM	*CORRECT ANSWER*	*SUBJECT'S ANSWER*	*TIME*
1.	B is not preceded by A - BA	True	~~F~~	8.05
2.	B does not follow A - AB	False	~~F~~	4.05
3.	B follows A - AB	True	~~F~~	2.67
4.	B precedes A - AB	False	F	2.09
5.	A is followed by B - AB	True	T	2.19
6.	B is not followed by A - BA	False	F	3.25
7.	A does not precede B - BA	True	T	3.18
8.	B is preceded by A - AB	True	T	2.49
		TOTAL	8	

T^{Ear} T^{R}

~ 2 min.

MISC.36/1

AB REASONING TEST

SUBJECT'S NAME: .....

DATE: .....

PRE-IMMERSION

	ITEM	*CORRECT ANSWER*	*SUBJECT'S ANSWER*	*TIME*
1.	A is not followed by B - AB	False	F	3.08
2.	A does not precede B - AB	False	F	3.47
3.	A precedes B - AB	True	T	2.14
4.	B does not follow A - BA	True	T	3.43
5.	B is not preceded by A - AB	False	F	2.34
6.	B is followed by A - AB	False	F	3.13
7.	A follows B - BA	True	T	2.42
8.	A is preceded by B - BA	True	T	3.48.
		TOTAL		

T^{Ear} T^{R}

1½ - 2 min

AB REASONING TEST

SUBJECT'S NAME: .....

DATE: .....

IMMEDIATE IMMERSION

	ITEM	*CORRECT ANSWER*	*SUBJECT'S ANSWER*	*TIME*
1.	B follows A - BA	False		
2.	B is preceded by A - BA	False		
3.	A precedes B - BA	False		
4.	B is not followed by A - AB	True		
5.	A is not preceded by B - AB	True		
6.	A does not follow B - BA	False		
7.	B does not precede A - AB	True		
8.	A is followed by B - BA	False		
		TOTAL		

T^{Ear} T^{R}

AB REASONING TEST

SUBJECT'S NAME: .....

DATE: .....

TEST SERIES: IMMERSED - end of run 12.07

ITEM	*CORRECT ANSWER*	*SUBJECT'S ANSWER*	*TIME*
1. B precedes A - BA	True	✓	1.93
2. B is followed by A - BA	True	✓	2.56
3. A does not follow B - AB	True	✓	2.15
4. A follows B - AB	False	✓	1.69
5. B does not precede A - BA	False	✓	2.44
6. A is not followed by B - BA	True	✓	2.66
7. A is not preceded by B - BA	False	✓	6.90
8. A is preceded by B - AB	False	✓	3.15
	TOTAL		

T^{Ear} T^{R}

MISC.36/4

AB REASONING TEST

SUBJECT'S NAME: .....

DATE: .....

REWARMING 3 min from start

ITEM	*CORRECT ANSWER*	*SUBJECT'S ANSWER*	*TIME*
1. A is followed by B - BA	False		
2. A precedes B - BA	False		
3. B does not precede A - AB	True		
4. B is not preceded by A - AB	False		
5. A does not follow B - BA	False		
6. B follows A - AB	True		
7. B is preceded by A - AB	True		
8. A is not followed by B - AB	False		
	TOTAL		

T^{Ear} T^{R}

AB REASONING TEST

SUBJECT'S NAME: .....

DATE: .....

REWARMING Final

	ITEM	*CORRECT ANSWER*	*SUBJECT'S ANSWER*	*TIME*
1.	B is preceded by A - BA	False	✓	8·55
2.	B is not followed by A - AB	True	✓	3 41
3.	B is followed by A - BA	True	✓	2·68
4.	B precedes A - BA	True	✓	1·66
5.	A is not preceded by B - BA	False	✓	2·72
6.	B does not follow A - BA	True	✓	3·08
7.	A follows B - BA	True	✓	2·20
8.	A does not precede B - BA	True	✓	3·10
		TOTAL		

T^{Ear} T^{R}

APPENDIX FIVE

—oOOoo—

MATERIAL INCLUDED IN THE THESIS WHICH HAS ALREADY BEEN PUBLISHED

Restoration of thermoregulatory response to body cooling by cooling hands and feet

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VAN SOMEREN, R. N. M., S. R. K. COLESHAW, P. J. MINCER, AND W. R. KEATINGE. *Restoration of thermoregulatory response to body cooling by cooling hands and feet.* J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 53(5): 1228–1233, 1982.—Deep body temperature fell progressively by 0.5–1.4°C during 3-h immersions in 29°C water. Both in unacclimatized volunteers and, to a lesser degree, in divers in cold-water training, cooling the hands and feet for 1 h in 12°C water during such immersion caused sensation of cold, shivering, and rise in metabolic rate; it caused body temperature to rise in unacclimatized subjects and halted its fall in divers. Tissue conductances generally fell a little in divers but rose in unacclimatized subjects, probably because of muscle blood flow associated with the greater shivering in the latter. Soaking the skin for 4 h produced no major changes in cutaneous thermal sensation assessed in the forearm, though with seawater it sometimes reduced cold sensation and with distilled water sometimes reduced warm sensation, a little. It is concluded that uniform skin temperature of 29°C often induces insufficient heat-gain reflexes to maintain body temperature and that cooling of the extremities can restore adequate thermoregulatory response.

thermoregulation; hypothermia; acclimatization; diving; metabolic response to cold; vasoconstrictor response to cold

HYPOTHERMIA, with deep body temperatures below 35°C, has been reported during diving in the North Sea using the conventional warm water flooding system (23) and during a laboratory immersion in water at 29°C (14). In both situations, cooling was insidious with little shivering or sensation of cold.

The present experiments were designed to see whether insidious body cooling of this kind resulted either from exposure to a uniform skin temperature near 29°C or from some effect of prolonged immersion of the skin on local temperature receptors. They were also designed to see whether local cooling of the hands and feet could restore normal thermoregulatory response; the hands and feet are normally cooler than skin of the trunk in air, but not during immersions in lukewarm water or during use of the warm water flooding system in deep dives. As regards the effect of immersion on receptors, calcium ions are known to depress the response of cutaneous cold receptors (18) and are present in high concentration in seawater, but it is not known whether they or any other ions penetrate from the skin surface in sufficient quantity to affect the receptors significantly. Similarly, although warming of the face is known to generate heat-loss reflexes (1), there is little information about the role of cold receptors in the hands and feet in generating the heat-gain reflexes required for thermoregulation in a cool environment.

In the first series of experiments, volunteers without recent experience of cold exposure and amateur shallow-water divers who had been training in cold water using unheated suits were immersed in water at 29°C. Their hands and feet were then cooled while the rest of the limbs and trunk remained in water at 29°C. Body temperature, metabolic rate, and regional heat losses were measured during this immersion. In the second series of experiments, one forearm was immersed in either distilled water or seawater for 4 h and the thermal sensation in it compared at intervals with that of the opposite forearm. When the sensations differed, the temperature of water around the test arm was varied until it felt the same temperature as the control arm.

METHODS

Immersions. The subjects for the main immersion experiments were 14 male and 5 female volunteers, aged 19–43. Six were amateur Subaqua divers who had dived for approximately 1.5 h/wk wearing unheated suits in water at 2–12°C during the 3 mo before the experiments. The other 13 subjects were indoor workers who had had no cold-water experience or other unusual cold exposure during the 3 mo before the experiments. Before immersion, all subjects were medically examined, their heights and weights were measured, and fat thickness was measured ultrasonically (Wells Krautkramer model USM 2F) at four sites: front of middle of upper arm; anterolateral point of middle third of trunk; 20 mm to the side of the midline, middle of lower third of trunk; and anterior midline of the middle of the thigh. Mean fat thickness was calculated from the sum (S) of these measurements in mm, as $1.308 + 0.181 \times S$ for men, and $1.933 + 0.168 \times S$ for women (15). Surface area was calculated from height and weight (8).

The subjects were fitted with electrocardiograph (ECG) leads on the right shoulder and the fifth left intercostal space in the midclavicular line; ECG was displayed on a monitor throughout the experiment. A digital voltmeter (Fenlow Electronics, type 105, input impedance 500 MΩ) recorded the output from heat-flow bands, which we have described previously and which are calibrated to allow for the local reduction in heat loss

that they themselves produce (12). These bands were placed on the right side of the body around the hand, foot, forearm, calf, and posterior and lateral aspects of the middle third of the trunk. Tissue conductance to each region of the body surface was calculated as the rate of heat loss measured locally by the heat-flow band, divided by the difference between deep body temperature and external water temperature. A zero-gradient aural thermometer (24), in which enough heating is supplied to the outer ear to eliminate local temperature gradients, was placed in the right external auditory meatus to measure deep body temperature. Metabolic rate was calculated by the method of Weir (31), from the oxygen deficit and volume at STP of expired air collected for 3- to 6-min periods, via a mouthpiece and valves. Oxygen content was measured by a paramagnetic analyzer (Beckman model E2) and volume by a dry gas meter (Gallenkamp model GF 095).

After initial measurements were made, the subjects were immersed in water to the neck, male subjects wearing only bathing trunks and female subjects a two-piece bathing suit. They sat on a slatted seat. The water was stirred by compressed air driven through three ducts, which directed streams of water onto the subject at approximately 1 $m \cdot s^{-1}$ from different angles. Air temperature was 22–24°C.

In the control experiments, six unacclimatized volunteers were immersed in water at 29 ± 0.1°C for 3 h. In the next experiments, 12 volunteers, 6 of whom were divers, were immersed in water at 29 ± 0.1°C in the same manner, but after 70–90 min, when heat loss from the hands and feet had stabilized so that valid estimations of regional conductance could be made, cold water at 12 ± 1°C was circulated through boxes that enclosed their hands and feet. The change in temperature in the box was complete (12 ± 1°C) within 3–5 min of the start of cooling. Latex seals at the wrists and ankles prevented movement of water between the boxes and the tank; care was taken to ensure that the seals did not restrict blood flow. After heat loss from the hands and feet had again stabilized, they were returned to water at 29 ± 0.1°C. Additional experiments of a similar kind were performed to see the effect of milder cooling of the hands and feet, first in water at 25 ± 0.1°C and then at 20 ± 0.1°C, with each of two subjects. Following cold immersions, subjects were rewarmed in a bath at 43 ± 1.0°C until deep body temperature rose to preimmersion levels.

Thermal sensation in the forearm. Fifteen men and nine women aged 19–29 yr, none of whom had had any recent exposure to unusual cold, acted as subjects. For each experiment, the flexor surface of one (right) forearm was immersed in a bath of stirred water at 15, 22, 29, or 38°C for 4 h, while the opposite (control) forearm was exposed to air at a temperature that kept its flexor surface temperature the same (±1.0°C) as that of the test forearm. Skin temperature of each forearm was measured by end-to-end insulated copper-constantan thermocouples, with a 99% response time of 1.5 s. The sensing point of each was held pressed to the skin but was not covered. At intervals, the flexor surface of the control forearm was immersed in a second water bath at the same temperature as that of the test water bath, and after 3 min the subject compared the thermal sensation from the two forearms. If the sensations differed, the temperature of the test bath was altered until the thermal sensations were equal at a time when skin temperature had been stable for at least 3 min. The temperatures of the two forearms were then noted.

The entire experiment was performed with the test forearm soaked in distilled water at each of four temperatures, 15, 22, 29, and 38°C and again at each of these temperatures with seawater around the test arm. The subjects were not informed about the actual temperatures of the test and the control arms during the experiment.

Statistical comparisons. Comparisons of sets of data were made by the *t* test. When comparing results from the same subjects in different conditions, values from given subjects were paired.

RESULTS

Immersion in water at 29°C. Six subjects were immersed for 3 h in water at 29°C. They were four men and two women aged 22–29 yr [mean 25 ± 1.4 (SE) yr]. Their height was 1.76 ± 0.05 m, weight 72.7 ± 4.0 kg, and mean overall fat thickness 8.7 ± 0.86 mm. All of them were indoor workers and nondivers with no unusual recent exposure to cold.

Figure 1 shows that their deep body temperature fell throughout the immersion, but metabolic rate rose little. The average fall of temperature over the 3-h period was 0.9 ± 0.17°C, the smallest fall being 0.5 and the largest 1.4°C. The lowest body temperature reached in any subject was 35.6°C. Falls in temperature were generally larger in the thinner subjects; correlation coefficients between fall in temperature and inverse of the individual's mean subcutaneous fat thickness were 0.88 for the four male subjects and 0.71 for the four male and two

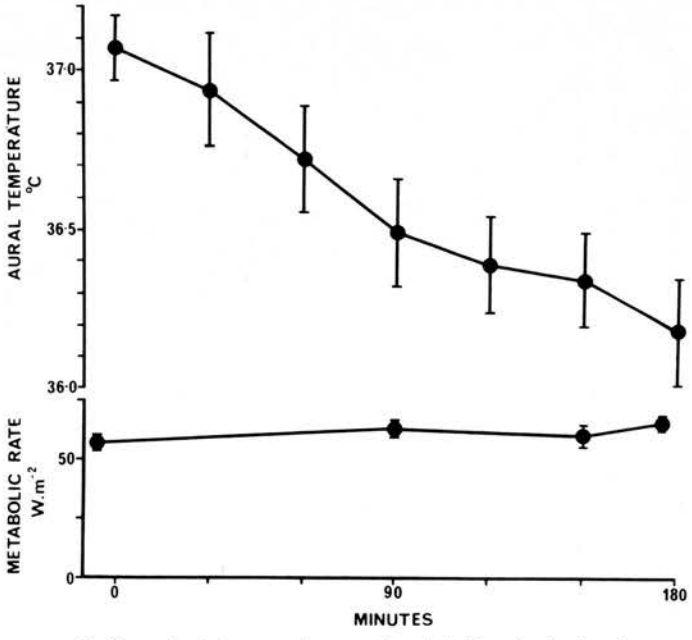

FIG. 1. Deep body temperature and metabolic rate during immersion in water at 29°C. Values are means ± SE of experiments on 6 subjects, none of whom had recently been exposed to cold.

female subjects together but did not reach statistical significance at the 0.05 level with these subjects alone. Metabolic rate with the subject sitting quietly before immersion was 50.7 ± 3.3 $W \cdot m^{-2}$; it rose only slightly to 60.6 ± 3.4 $W \cdot m^{-2}$ 90 min after the start of the immersion, and to 65.4 ± 2.8 $W \cdot m^{-2}$ just before the end of the immersion. The subjects shivered only slightly and intermittently during the immersion and reported little sensation of cold.

Figure 2 shows that during the first 30–60 min of immersion regional heat losses were at high levels, which were largely attributable to loss of stored heat from peripheral tissues. Heat losses then became fairly stable at levels and indicated tissue conductances that were substantially lower in the limbs than in the trunk.

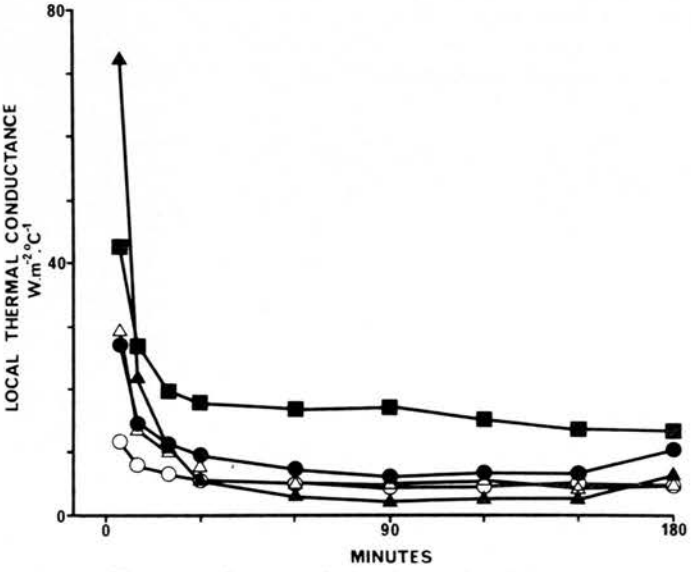

FIG. 2. Tissue conductance during immersion in water at 29°C. Values are means ± SE for same 6 subjects as in Fig. 1. ■, Trunk; ▲, hand; ●, forearm; △, foot; ○, calf. SEs at 180 min ($W \cdot m^{-2} \cdot °C^{-1}$): trunk, ±4.3; hand, ±1.5; forearm, ±1.7; foot, ±0.9; calf, ±0.3.

Effect of cooling hands and feet during general immersion at 29°C. Six divers and six nondivers were immersed to the neck in water at 29°C. After 70–90 min, when regional heat losses were stable, their hands and feet were cooled in water at 12°C. The divers included one woman and the nondivers two women. The divers were aged 21–43 yr (mean ± SE, 30 ± 3.6) compared with 19–28 yr (24 ± 1.4) for the nondivers and were on average a little taller (1.76 ± 0.04 m) than the nondivers (1.71 ± 0.04 m), heavier (72.7 ± 3.0 compared with 64.4 ± 4.8 kg), and had a rather larger mean subcutaneous fat thickness (8.2 ± 1.09 compared with 6.7 ± 0.62 mm). None of the differences between the two groups was statistically significant.

Figure 3 (top) shows that, in water at 29°C, the deep body temperatures of the divers and nondivers fell at similar mean rates of approximately $0.4°C \cdot h^{-1}$. Again, rates of fall were faster in thinner than fatter subjects among the nondivers; correlation coefficients between rate of fall and the inverse of mean subcutaneous fat thickness were 0.78 for the male nondivers and 0.66 for all of the nondivers during the first 70 min of immersion at 29°C. When results for the male nondivers and the male controls of the previous experiments (also nondivers) were combined for the first 70 min of immersion at 29°C (total 8 subjects), the relationship between fall of deep body temperature and inverse of mean subcutaneous fat thickness was significant ($P < 0.05$, $r = 0.70$). No significant relationship was found between fall of deep body temperature and subcutaneous fat thickness in divers; for the male divers $r = 0.33$, and for all of the divers $r = 0.03$.

When the hands and feet were cooled in water at 12°C after 70–90 min of immersion at 29°C, deep body temperature of the nondivers ceased to fall and then rose

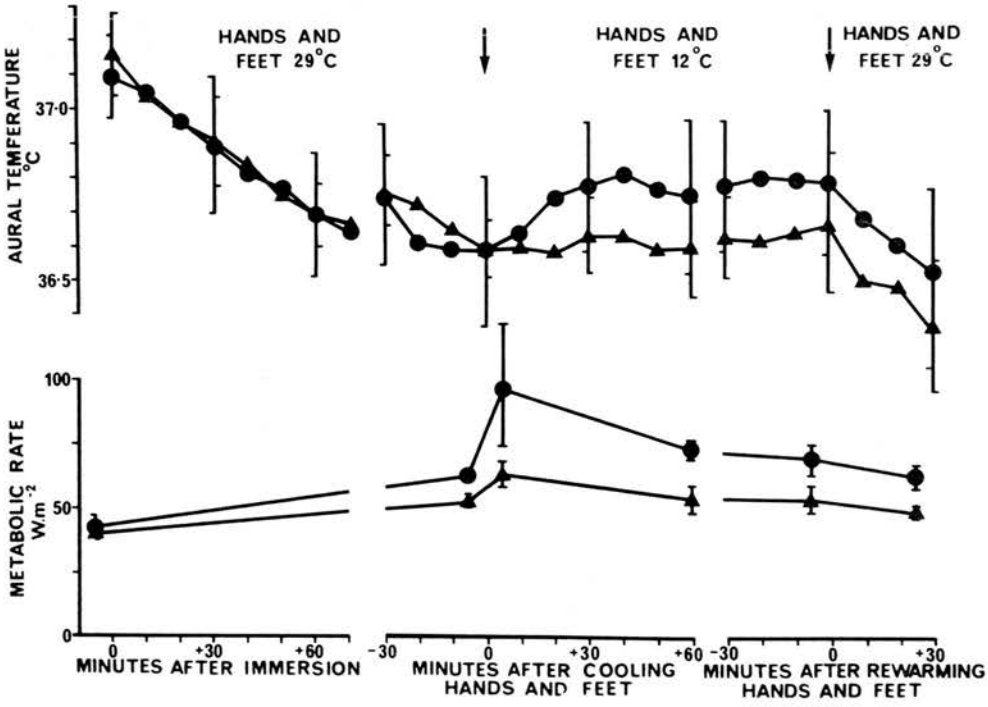

FIG. 3. Effect of cooling hands and feet in 12°C water, during general immersion in water at 29°C, on body temperature and metabolic rate. ● Means ± SE, 6 unacclimatized subjects; ▲ means ± SE, 6 divers in cold-water training.

slightly but significantly ($P < 0.05$) from 36.6 ± 0.2 to 36.8 ± 0.2°C by the end of 30 min; that of the divers ceased to fall but did not rise significantly. When the hands and feet were returned to water at 29°C, the falls in deep body temperature were resumed, at a rate of approximately $0.6°C \cdot h^{-1}$, in both divers and nondivers.

Before immersion, with the subjects sitting quietly, metabolic rates were similar in the divers and nondivers, 41.0 ± 2.5 and 42.9 ± 3.8 $W \cdot m^{-2}$, respectively (Fig. 3, bottom). After 70–90 min of immersion in water at 29°C, metabolic rates had risen significantly in both groups ($P < 0.05$) but by only 26% in the divers compared with 45% in the nondivers; there was little obvious shivering and little sensation of cold in either group. Metabolic rates were then significantly lower in the divers than the nondivers. Five minutes after the start of hand and foot cooling, the metabolic rate of the divers had risen to 54% above resting level and that of nondivers to as much as 125% above resting level. Both of these increases were significant ($P < 0.05$), and the metabolic rate of the divers remained significantly lower than that of the nondivers. The nondivers all shivered markedly during cooling of the hands and feet, while the divers shivered noticeably less. All subjects, but particularly the nondivers, complained of both local cold sensation in the hands and feet and a general feeling of cold at this time. Metabolic rate, shivering, and sensations of cold then declined in both groups while the hands and feet remained at 12°C, but in nondivers they remained significantly above the previous level throughout the time that the hands and feet were cooled. All of the divers subsequently volunteered the information that they had learned to suppress shivering deliberately during dives in cold water. The smaller metabolic responses of the divers compared with nondivers can therefore account for the failure of divers to increase body temperature during cooling of the hands and feet as nondivers did. It can also explain why divers initially cooled at similar rates to nondivers in water at 29°C, in spite of the divers' being generally fatter and presumably therefore having more tissue insulation than nondivers.

Figure 4 shows that tissue conductances did not alter greatly on cooling the hands and feet in water at 12°C or on returning them to water at 29°C. In general, tissue conductances tended to decline during cooling of the hands and feet in divers and to rise in the nondivers. The changes were small and generally not significant, but in divers conductance to the forearm was significantly less ($P < 0.05$) during cooling of the hands and feet than the mean conductance there before and after the cooling. The greater mean trunk insulation observed in divers throughout most of the experiment is attributable to their greater mean fat thickness; the lower mean limb insulations in divers compared with nondivers imply less vasoconstriction in the divers.

Figure 5 shows that less intense cooling of the hands and feet, in water at 25 and 20°C, still produced clear and sustained increases in deep body temperature, and usually in metabolic rate, in two subjects who were again otherwise immersed to the neck in water at 29°C. Both subjects were nondivers and had not experienced recent exposures to cold except that they had been subjects for the control immersions at 29°C. One was male, 29 yr, 1.83 m, 77.0 kg, with a mean subcutaneous fat thickness of 7.5 mm. The other was female, 22 yr, 1.63 m, 66.3 kg, with a mean subcutaneous fat thickness of 10.5 mm.

Effect of prolonged soaking of the skin on local thermal sensation. The differences in skin temperature needed to produce the same thermal sensation in test forearms that had been soaked in water, and in the opposite, control forearms, are shown in Table 1. Neither soaking the skin of the forearm in fresh water nor seawater for 3 h had an important effect on local sensation of heat or cold, although fresh water sometimes caused the skin to feel a little colder and seawater a little warmer than the control arm at the same temperature. In particular, soaking in distilled water for 2–3 h produced an apparent small decrease in warm sensation at 38°C, requiring the soaked forearm to be a little more than 0.2°C warmer than the control forearm to match its

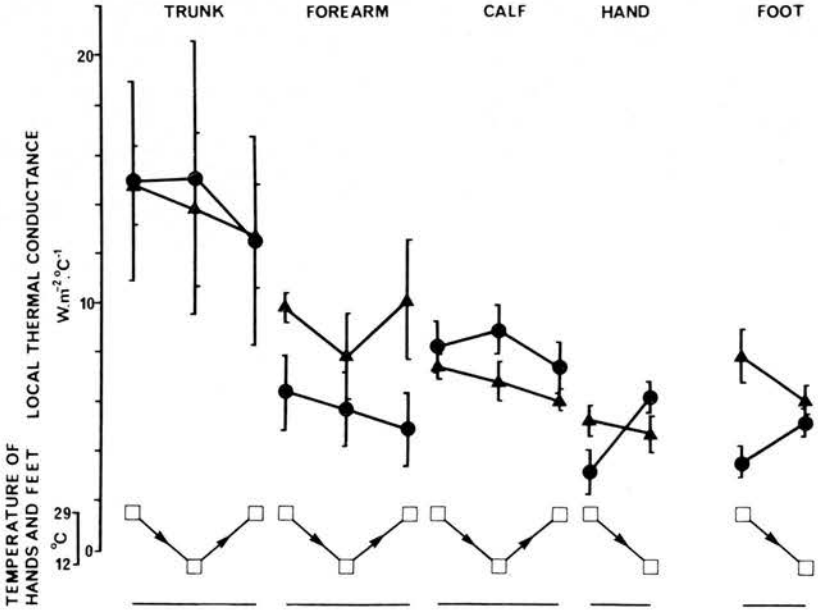

FIG. 4. Effect of cooling hands and feet in 12°C water, during general immersion in water at 29°, on tissue conductance. Symbols as in Fig. 3; same experiments on same subjects as in Fig. 3. Values are stable values reached at end of periods with hands and feet in water at 29°C, then 12°C, then 29°C. Stable values were not reached for hands and feet in short second exposure to 29°C.

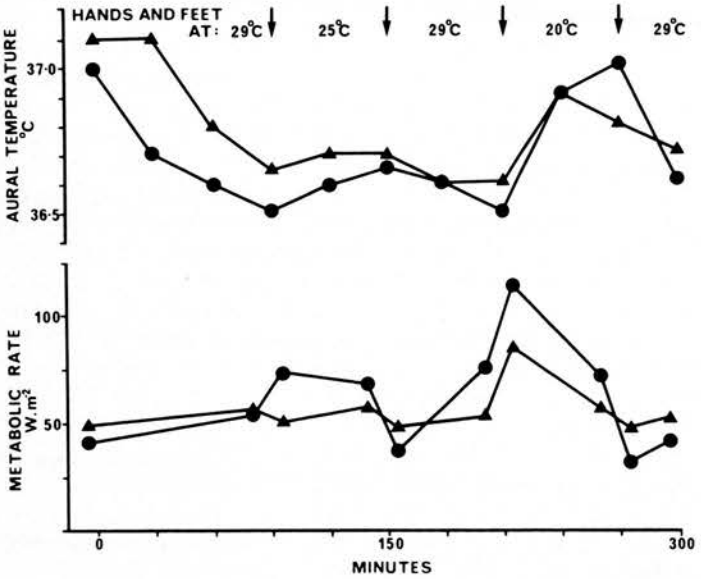

FIG. 5. Effect of cooling hands and feet in 25°C and 20°C water, during general immersion in water at 29°C, on deep body temperature and metabolic rate. Two subjects, both unacclimatized to cold: ●, male subject; ▲, female subject.

TABLE 1. *Effect of soaking skin on thermal sensation in forearm*

	Duration of Soaking, min	Surface Temperature of Control Forearm, °C			
		38	29	22	15
		°C differential for equal sensation in soaked forearm			
Soaking in seawater	60	0.00	−0.01	−0.20	0.15
		±0.07	±0.60	±0.10	±0.24
	120	−0.20	0.00	−0.01	0.18
		±0.19	±0.12	±0.06	±0.16
	180	−0.33	0.01	−0.28*	0.05
		±0.26	±0.12	±0.09	±0.28
Soaking in distilled water	60	0.05	−0.01	−0.04	0.04
		±0.06	±0.05	±0.09	±0.23
	120	0.22*	0.09	0.03	0.20
		±0.07	±0.08	±0.06	±0.12
	180	0.21	0.07	0.02	0.09
		±0.13	±0.06	±0.20	±0.11

Values are means ± SE for 6 experiments and represent surface temperature of test forearms producing the same sensation as control forearm, in °C, above that of the control forearm. * Significantly different from zero, $P < 0.05$.

sensation, whereas soaking in seawater for similar times produced a probable small increase of similar size in warm sensation at 38°C and decrease in cold sensation at 22°C.

DISCUSSION

Perhaps the most striking finding was that immersion in lukewarm water (29°C) induced in every subject a progressive fall in deep body temperature, which was accompanied by little shivering or sensation of cold, which continued throughout a 3-h immersion and which was as large as 1.4°C in one case. It is known that some input from cutaneous cold receptors is needed to allow falls in deep body temperature to induce a substantial metabolic response to cold, both in dogs (5) and humans (3, 26). Our findings imply that a uniform cutaneous temperature of 29°C often produced insufficient sensory input for an adequate thermoregulatory response even when deep body temperatures were as low as 35.6°C. This is at first sight surprising, since skin temperatures in the region of 25–29°C have generally been found to produce maximal rates of summated discharge from groups of cutaneous cold receptors (see Ref. 17 for review). In particular, cold receptors of the monkey hand produce maximal summated rates of discharge at 25°C (10) and only slightly lower rates at 29°C. Even after cold acclimatization, by several years' exposure to air at 5°C, the temperature producing maximum discharge of cold receptors in the cat's face was 25°C, and rates of discharge at 29°C were only a little below this maximum (19). However, individual cold receptors within the groups of receptors that generate these summated rates of discharge vary greatly with regard to the skin temperature at which they give a maximal response; some individual cold receptors in the cat's face give maximal discharges at a surface temperature as low as −5°C (9), whereas others do so at a surface temperature as high as 40°C (19). The fact that cutaneous temperatures of 25–29°C feel less cold than temperatures of 12°C (4) and also, in our experiments, generated lower reflex responses to cold might therefore result from receptors that respond maximally at 25–29°C, although more numerous, producing less sensation of cold and less activation of thermoregulatory reflexes than receptors that respond maximally at lower temperatures.

The fact that cooling the hands and feet produced a thermoregulatory response adequate to stabilize body temperature is surprising in view of the fact that these regions form only about 12% of the body surface area (8). The only indication of the density of cold receptors in hands and feet is provided by an observation (30) that "cold spots" were less dense there than in the skin of either the face or trunk. If this does reflect the density of cold receptors, the importance of the hands and feet in the sensory response to cold may lie mainly in the fact that in air they are particularly exposed to environmental temperature and that their input is accordingly particularly important in generating overall thermoregulatory responses. The face is also usually exposed, and warming the face is known to be more effective than warming the trunk in inducing a reflex increase in peripheral blood flow (1). In the cold, however, the head is a region of low internal insulation and high heat loss (11, 15) so that the skin there cools relatively little. The hands and feet are regions of low heat loss on exposure to cold (at least down to a temperature of 12°C, at which cold vasodilatation develops) largely because of efficient counter-current exchange in the limbs when blood flow is low (2). This normally results in the hands and feet being cooler than other parts of the skin, and it enables them to act as effective external cold sensors without serious local heat loss. A uniform skin temperature of 29°C, such as is produced by immersion in lukewarm water, is an unusual situation. Our results indicate that it is one in which the human thermoregulatory system is not normally adjusted to respond effectively.

An obvious practical conclusion of the present findings

is that diver heating systems, which allow the surface temperature of the hands and feet to fall several degrees below that of the trunk, are likely to reduce the risk of insidious hypothermia. Another practical implication is that active heating of gloves and footwear in either cold air or cold water may carry the risk of inducing insidious hypothermia. This particularly applies immediately after heating is started. A stepwise increase in the temperature of cutaneous cold receptors causes an initial large decrease and a smaller sustained decrease in their rate of firing, with converse effects on cooling (7, 20). Large early changes as well as smaller sustained changes in metabolic rate and body temperature, corresponding to these dynamic and static phases, were seen when the hands and feet of our subjects were cooled and again when they were heated.

Cold-acclimatized people tend to vasoconstrict and to shiver less in the cold and therefore increase metabolic rate less than warm-acclimatized people, although more complex patterns of change can occur with certain patterns of acclimatization, particularly when they involve periods of prolonged exposure to cold (6, 13, 21, 22, 25, 29). The reasons for this include the establishment of conditioned reflexes. In our subjects, however, the fact that divers in cold-water training all stated that they had learned to control shivering voluntarily suggests that voluntary control, learned to minimize use of breathing gas and to prevent shivering from impeding their activities in the water, was responsible for much of their low metabolic response compared with nondivers with similar levels of deep and surface temperature.

Since seawater is rich in calcium (20.4 meq·l^{-1}) (27), and since high calcium levels depress the discharge of cold receptors and increase that of warm receptors in the skin (18, 28), prolonged immersion in seawater might be expected to make the skin feel warmer at any given temperature. Fresh water, absorbed osmotically to dilute tissue electrolytes, might have the reverse effect. In practice, we could demonstrate only a small, and generally nonsignificant, tendency of the skin to feel warmer after soaking for up to 3 h in seawater, and to feel colder after such soaking in fresh water. The depth of the warm receptors is not known, but cutaneous cold receptors lie mainly in the dermis with a small part of their terminal regions penetrating the epidermis (16). It seems that, in spite of their rather superficial location, these receptors are only slightly affected by even prolonged changes in ionic composition at the surface of the epidermis.

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REFERENCES

1. BADER, M. E., AND M. B. MACHT. Indirect peripheral vasodilatation produced by the warming of various body areas. *J. Appl. Physiol.* 1: 215–226, 1948.
2. BAZETT, H. C., L. LOVE, M. NEWTON, L. EISENBERG, R. DAY, AND R. FORSTER. Temperature changes in blood flowing in arteries and veins in man. *J. Appl. Physiol.* 1: 3–19, 1948.
3. BENZINGER, T. H., A. W. PRATT, AND C. KITZINGER. The thermostatic control of human metabolic heat production. *Proc. Natl. Acad. Sci. USA* 47: 730–739, 1961.
4. BESTE, R. *Perzeption statischer thermischer Reize beim Menschen* (inaugural dissertation). Marburg, 1977. Quoted by: H. Hensel (17).
5. BRENDEL, W. Die Bedeutung des Hirntemperatur für die Kaltegegen-regulation. I and II. *Pfluegers Arch.* 270: 607–647, 1960.
6. CARLSON, L. D., H. L. BURNS, T. H. HOLMES, AND P. P. WEBB. Adaptive changes during exposure to cold. *J. Appl. Physiol.* 5: 672–676, 1953.
7. DOSTROVSKY, J. O., AND R. F. HELLON. The representation of facial temperature in the caudal trigeminal nucleus of the cat. *J. Physiol. London* 277: 29–47, 1978.
8. DUBOIS, D., AND E. F. DUBOIS. Clinical calorimetry: a formula to estimate the approximate surface area if height and weight be known. *Arch. Intern. Med.* 17: 863–871, 1916.
9. DUCLAUX, R., K. SCHAFER, AND H. HENSEL. Response of cold receptors to low skin temperatures in nose of the cat. *J. Neurophysiol.* 43: 1571–1577, 1980.
10. DYKES, R. W. Coding of steady and transient temperatures by cutaneous "cold" fibres serving the hand of monkeys. *Brain Res.* 98: 485–500, 1975.
11. FROESE, G., AND A. C. BURTON. Heat losses from the human head. *J. Appl. Physiol.* 10: 235–241, 1957.
12. GIN, A. R., M. G. HAYWARD, AND W. R. KEATINGE. Method for measuring regional heat losses in man. *J. Appl. Physiol.: Respirat. Environ. Exercise Physiol.* 49: 533–535, 1980.
13. HANNA, J. M., AND S. K. HONG. Critical water temperature and effective insulation in scuba divers in Hawaii. *J. Appl. Physiol.* 33: 770–773, 1972.
14. HAYWARD, M. G., AND W. R. KEATINGE. Progressive symptomless hypothermia in water: possible cause of diving accidents. *Br. Med. J.* 1: 1182, 1979.
15. HAYWARD, M. G., AND W. R. KEATINGE. Roles of subcutaneous fat and thermoregulatory reflexes in determining ability to stabilise body temperature in water. *J. Physiol. London* 320: 229–251, 1981.
16. HENSEL, H. Thermoreceptors. *Annu. Rev. Physiol.* 36: 233–249, 1974.
17. HENSEL, H. *Thermoreception and Temperature Regulation.* London: Academic, 1981, p. 43–51.
18. HENSEL, H., AND K. SCHAFER. Effects of calcium on warm and cold receptors. *Pfluegers Arch.* 352: 87–90, 1974.
19. HENSEL, H., AND K. SCHAFER. Activity of cold receptors in cats after longterm adaptation to various temperatures (Abstract). *Pfluegers Arch.* 379: R56, 1979.
20. HENSEL, H., AND Y. ZOTTERMAN. The response of the cold receptors to constant cooling. *Acta Physiol. Scand.* 22: 96–113, 1951.
21. KEATINGE, W. R. The effect of repeated daily exposure to cold and of improved physical fitness on the metabolic and vascular response to cold air. *J. Physiol. London* 157: 209–220, 1961.
22. KEATINGE, W. R., AND M. EVANS. Modification of acute reflex responses to cold by brief training in a cold climate. *Lancet* 2: 1038–1041, 1958.
23. KEATINGE, W. R., M. G. HAYWARD, AND N. K. I. MCIVER. Hypothermia during saturation diving in the North Sea. *Br. Med. J.* 1: 291, 1980.
24. KEATINGE, W. R., AND R. E. G. SLOAN. Deep body temperature from aural canal with servo-controlled heating to outer ear. *J. Appl. Physiol.* 38: 919–921, 1975.
25. LE BLANC, J. Evidence and meaning of acclimatization to cold in man. *J. Appl. Physiol.* 9: 395–398, 1956.
26. NIELSON, B. Metabolic reactions to changes in core and skin temperature in man. *Acta Physiol. Scand.* 97: 129–138, 1976.
27. PROSSER, C. L. *Comparative Animal Physiology.* Philadelphia, PA: Saunders, 1973, p. 84.
28. SCHAFER, K., H. A. BRAUN, AND H. HENSEL. Dependence of cold fibre response from blood calcium level in the cat (Abstract). *Pfluegers Arch.* 373: R68, 1978.
29. SKRESLET, S., AND F. AAREFJORD. Acclimatization to cold in man induced by frequent scuba diving in cold water. *J. Appl. Physiol.* 24: 177–181, 1968.
30. STRUGHOLD, H., AND R. PORTZ. Die Dichte der Kaltpunkte auf der Haut des menschlichen Korpers. *Z. Biol.* 91: 563–571, 1931.
31. WEIR, J. B. DEV. New methods for calculating metabolic rate with special reference to protein metabolism. *J. Physiol. London* 109: 1–9, 1949.

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Thermoregulatory response to cooling of hands and feet

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Immersion in lukewarm water at 29 °C can lead to a progressive fall in deep-body temperature with little metabolic response or awareness of cold (Hayward & Keatinge, 1979). Similar insidious hypothermia can occur in North Sea divers using the warm-water flooding system (Keatinge & Hayward, 1980). We have now investigated the effect of local cooling of the hands and feet on body cooling in such conditions.

Deep-body temperature of volunteers was measured by a zero-gradient aural thermometer (Keatinge & Sloan, 1975) during immersion to the neck in stirred water at 29 °C. After 75–90 min, when their body temperatures had fallen from a mean of 37·1 to 36·6 °C, cold water at 12 ± 1 °C was circulated through boxes surrounding their hands and feet. Deep body temperature ceased to fall, and on average rose slightly to 36·7 °C during the next 60–70 min. Returning the hands and feet to water at 29 °C was followed by resumption of body cooling.

The rise in body temperature during cooling of the hands and feet was associated with a sensation of cold, shivering, and an increase in metabolic rate. Heat loss from the hands and feet increased, but represented only a small proportion of overall heat loss from the body. Heat loss from other parts of the body altered little, probably because an increase in internal insulation due to vasoconstriction in the skin was balanced by a decrease in insulation due to a rise of blood flow in muscle, associated with shivering. The sensation of cold, shivering and increased metabolic rate ceased when the hands and feet were returned to water at 29 °C.

The results suggest that insidious hypothermia during uniform immersion of the skin in lukewarm water is due largely to lack of the sensory input from cutaneous cold receptors of the hands and feet which is normally present in air. Without this input, even large falls in deep body temperature often failed to evoke an adequate thermoregulatory response or sensation of cold. Cooling the hands and feet was sufficient to restore this response without greatly increasing heat loss from them, probably because of vasoconstriction and the countercurrent cooling of blood which is known to limit loss of heat from the extremities in the cold.

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REFERENCES

Hayward, M. G. & Keatinge, W. R. (1979). *Br. med. J.* **i**, 1182.
Keatinge, W. R. & Hayward, M. G. (1980). *Br. med. J.* **i**, 291.
Keatinge, W. R. & Sloan, R. E. G. (1975). *J. appl. Physiol.* **38**, 919–921.

Impaired memory registration and speed of reasoning caused by low body temperature

S. R. K. COLESHAW, R. N. M. VAN SOMEREN, A. H. WOLFF,
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COLESHAW, S. R. K., R. N. M. VAN SOMEREN, A. H. WOLFF, H. M. DAVIS, AND W. R. KEATINGE. *Impaired memory registration and speed of reasoning caused by low body temperature.* J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 55(1): 27–31, 1983.—Volunteers' body core temperatures were lowered by immersion in water at 15°C. Aspects of cognitive function were subsequently tested after rewarming had been started in water at 41°C when their skin was warm and they felt comfortable but their body core temperature remained low. Memory registration was found to be impaired progressively when core temperature fell from about 36.7°C; at core temperatures of 34–35°C the impairment caused loss of approximately 70% of data that could normally be retained. However, recall of previously learned data was not impaired at these core temperatures. On a two-digit calculation test, speed of performance was impaired by about 50% at a core temperature of 34–35°C, but provided enough time was available, accuracy of performance was not reduced.

cognitive function

IT HAS LONG BEEN KNOWN that severe hypothermia causes confusion and unconsciousness (6), though some degree of consciousness may be retained at body temperatures as low as 27°C (e.g., 14). The possibility that comparatively small depressions of body temperature might produce significant impairments of mental function became of particular interest in view of findings that core temperatures of North Sea divers were sometimes as low as 34.7–35.0°C at the end of dives, even when the warm-water flooding system of heating was in use (12). Similar levels of body temperature could sometimes be produced by mild uniform external cooling in lukewarm water at 29°C (5, 9, 18) and by recreational cold swims (15). Evidence that memory can be seriously affected by rapid cooling of the body core was provided by complete amnesia reported by two subjects for the last few minutes of 20-min immersions in water at 5°C, during which they cooled to core temperatures of 34.2 and 35.1°C (10). The possibility that discomfort due to the low cutaneous temperature played a part was not excluded in that study and was thought to be the cause of minor impairments in mental function observed during exposures to cold in later studies. In particular, Bowen (3) and Stang and Weiner (16) reported lengthening of the time needed to perform mental tasks when people, wearing foam-rubber wet-suits, were in cold water at 6 and 10°C for approximately 30 and 90 min, respectively. It was suggested that this was caused by the distracting effect of cutaneous cooling; deep body temperatures were uncertain. Baddeley and his colleagues (2, 7) noted impairments of cognitive processing in divers cooled in water to produce mean decreases in rectal temperature of 0.7 and 1.1°C, respectively. The impairments were again attributed to the distracting effects of the cold water, although it was noted in the second of these papers that word recognition was more impaired in subjects who had undergone relatively large decreases in central body temperature. We have now assessed memory registration and recall, and we have measured the accuracy and time needed for performance of reasoning tasks during mild depression of core temperature at a time when skin temperature was high enough to eliminate a sensation of cold discomfort.

METHODS

Subjects and procedure. Our subjects were 27 male and 10 female volunteers aged 20–40 yr. All were indoor workers, and none of them had undergone any unusual exposure to cold in the previous 3 mo. The subjects were immersed individually to the neck in a 4,000-liter tank of stirred water at 15 ± 0.2°C, but with their hands in sealed boxes of water at 29 ± 0.2°C to avoid impairment of manual dexterity. The immersions were planned to last 1 h but were discontinued if body temperature fell below 34.5°C or if the subject requested termination. Immersions of less than 45 min were not included in the results. At the end of this time they entered a warm bath at 41 ± 0.2°C and remained in it for 30–45 min until body core temperature had risen to within 0.3°C of its level before immersion in the cold water. Air temperature was 22–24°C. Fourteen of the subjects immersed were asked to memorize a test passage, starting approximately 3 min after entering the warm water. They were then asked to recall it 1 h later, after rewarming was complete and while they were resting and clothed, and in air at 21–23°C. Another 19 of the subjects were asked to memorize a test passage just before the cold immersion and then to recall it 1 h later, approximately 3 min after leaving the cold and entering the warm water. All of these subjects were also given a similar passage to learn and recall 1 h later, but under control conditions at normal body temperatures, clothed, and in air at 21–23°C during both learning and recall. These control tests were made 1–7 days before or after the immersions.

Sixteen of the subjects who performed the memory

tests and one other (15 men and 2 women) also performed the calculation and reasoning tests. Each was given one series of these tests 1 h before immersion, another a few minutes before immersion, and two series of tests during the cold immersion, for training. They were then given another series starting approximately 6 min after leaving the water at 15°C and entering the bath at 41°C, and a final series while still in the bath at 41°C but after core temperature had returned to within 0.3°C of its level before the cold immersion. Results of these latter two series were then compared.

Memory tests. Two passages of the type devised by Friedman and Greitzer (8), each including 15 facts, were used. One passage was: "Around a small island, there are a number of boats that have been wrecked. There are three which are well known to divers. The first is a fishing boat called the Lady Lucy which lies in three metres of water amongst kelp; the only danger is the number of poisonous fish. The second, which is surrounded by sharks, is a battleship, HMS Intrepid, which sank in 20 metres of water and lies on a shingly bottom. The third is a coaster, called The Mermaid, which lies on a rocky bottom in 50 metres of water, but is now only a tangle of metal with very sharp edges."

The second passage was similar to the first, but the details were changed. The subject was allowed 2.5 min to learn the passage, which was written on a waterproof card, and was told when 1 min remained. To test recall the subject was asked to repeat as much of the passage as he could remember. The number of facts that were correctly stated was then noted.

Calculation and reasoning tests. There were three tests. The first consisted of 10 items each involving addition of 5 single digits (e.g., 7 + 6 + 8 + 9 + 5 =), the second consisted of 6 items each involving addition of 4 double digits (e.g., 72 + 21 + 94 + 55 =), and the third consisted of 8 items each involving a reasoning problem of the type devised by Baddeley (1) (e.g., A does not precede B; AB), the answer being either true or false. No problem was repeated during the experiment.

The subject held a bicycle grip and lever in each hand. The levers were connected mechanically by cables to an electronic digital clock (accurate to 0.01 s). Each problem was projected singly onto a screen 1.3 m wide, 2 m in front of the subject. The electronic clock started automatically as the slide was presented. For each calculation problem, the subject pressed the right-hand lever as soon as he reached a solution to record response time automatically and then called out his solution, which was noted by the observer as right or wrong. For A-B reasoning problems, the subject pulled the right-hand lever if he thought the statement was true or the left if he thought it was false. Lights corresponding to each lever were then illuminated on a panel in front of the experimenter, and the clock automatically stopped. In each case, the experimenter recorded the subject's answer and the time on the clock before resetting the clock to zero and projecting the next slide.

The subjects were unable to see the clock, and no feedback of performance was given by the experimenter.

Body core temperature. Deep body temperature was measured by a zero-gradient aural thermometer (13) that records temperature from the external auditory meatus at a depth of 10 mm. Local cooling from outside is prevented by a heating pad over the outer ear that is servo-controlled to keep its temperature the same as that being recorded from the meatus. With external air temperature of 22–24°C, as was present in these experiments, this method gives values within 0.3°C of the temperature recorded in the lower third of the esophagus even when core temperature is changing by 0.5°C in 10 min.

Fat thickness. Fat thickness was measured by an ultrasonic apparatus (Wells Krautkramer model USM 2F) at the following sites: front of middle of upper arm; anterolateral point of middle third of trunk; 20 mm to the side of the midline, middle of lower third of trunk; and anterior midline of the middle of the thigh. Mean fat thickness over the whole body (in mm) was calculated (18) from the sum of these readings (S) as 1.308 + 0.181 S in men and 1.933 + 0.168 S in women.

Statistical analysis. Significances were assessed using the *t* test with pairing when appropriate.

RESULTS

Figure 1 shows the changes in body temperature during a typical immersion at 15°C for 1 h, then at 41°C, and

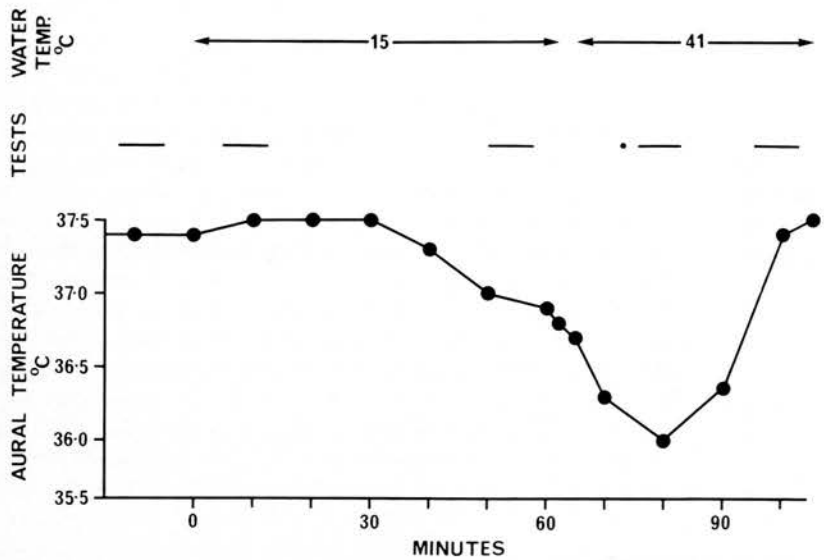

FIG. 1. Timing of tests before and during cold and warm immersion with change in core temperature of one subject. ——, Calculation and reasoning tests; final 2, in 41°C water, provided data analyzed in the text; the first, 1 h before immersion, is not shown. ·, Memory test; memorization period in subjects for Fig. 2, who were asked to recall facts learned 70–80 min later when they were rewarmed and in air; recall period in subjects for Fig. 3, who had memorized the facts 70–80 min earlier, in air, shortly before immersion.

the times at which tests of memory, calculation, and reasoning were made. The extent of the fall in body temperature after 1 h in the cold water (T, °C) varied from −0.2 to 3.3°C between subjects, mainly due to variations in their mean subcutaneous fat thickness (F, mm) which ranged from 3.9 to 11.8 mm. A clear inverse relationship, $T = (16.6/F) - 1.6$; $r = 0.71$, $P < 0.001$, was found between the rate of fall of temperature in the cold water and the mean subcutaneous fat thickness of each of the 37 subjects (11). The size of the afterdrop in temperature at the start of rewarming was often substantial, as in the case illustrated, and varied from 0.25 to 0.85°C. The size of the afterdrop (T, °C) was directly related to mean subcutaneous fat thickness ($T = 0.04F + 0.21$, $r = 0.44$, $P < 0.02$). The results in Fig. 1 are from a subject of near average fatness (mean fat thickness 7.0 mm) with a moderate fall of core temperature in cold water and also a moderate afterdrop.

Memory registration at reduced deep body temperature. Figure 2 shows the number of facts recalled by 14 subjects at normal body temperature, in air, after they had attempted to learn the passage at reduced body temperature. The attempt to learn the passage was made a few minutes after they left the cold for the warm bath, when shivering and discomfort had ceased. The facts recalled are expressed as a difference from the number of facts recalled by the subject during a similar test at normal body temperature throughout. There is a clear relationship ($r = 0.77$, $P < 0.002$) between performance and the level of deep body temperature at the time the passage was learned. With temperatures below about 36.7°C, there was progressive impairment which reached approximately 70% when the passage was learned at a temperature of 34–35°C. Memory score for the group as a whole was 8.6 ± 1.1 facts out of 15, and mean body temperature at the time they were learned was 35.6 ± 0.2°C (mean ± SE).

During the control tests on these subjects in air which were made 1–7 days before the immersion at normal body temperature throughout, memory score was 12.5 ± 0.5 facts out of 15. Body temperature in these conditions was 37.3 ± 0.1°C, and there was no significant correlation between this body temperature and control memory score ($r = 0.09$). Also there was no significant correlation between the memory scores under these two conditions ($r = 0.17$) or between the memory score of each subject in the control situation and his body temperature at the time he attempted to learn the similar test passage at reduced body temperature ($r = 0.19$).

Memory recall at reduced deep body temperature. Figure 3 shows the number of facts recalled by 19 subjects at reduced body temperature after they had learned a test passage at normal body temperature, again expressed as a difference from the number of facts recalled by the subject during a similar test at normal body temperature throughout. There is no tendency to reduced performance when recall was tested at body temperatures as low as 34.6°C. Memory score for the group was 11.3 ± 0.6 facts out of 15, and mean body temperature at the time they were recalled was 36.0 ± 0.2°C.

During the control tests on these subjects, in air, and at normal body temperature throughout, score for memory recall was 12.0 ± 0.7 facts out of 15. Body temperature in these conditions was 37.2 ± 0.6°C and there was then no significant correlation between this body temperature and control memory score ($r = -0.3$). Nor was there a significant correlation between the memory score of each subject in the control situation and his body temperature at the time he attempted to recall the similar test passage at reduced body temperature ($r = 0.02$), but there was a clear relationship between the memory scores under these two conditions ($r = 0.71$, $P < 0.001$). It can also be seen in Fig. 3 that there is no systematic difference in performance at a given body temperature between those subjects who performed the control test of recall before and those who performed it after the test of recall at reduced body temperature.

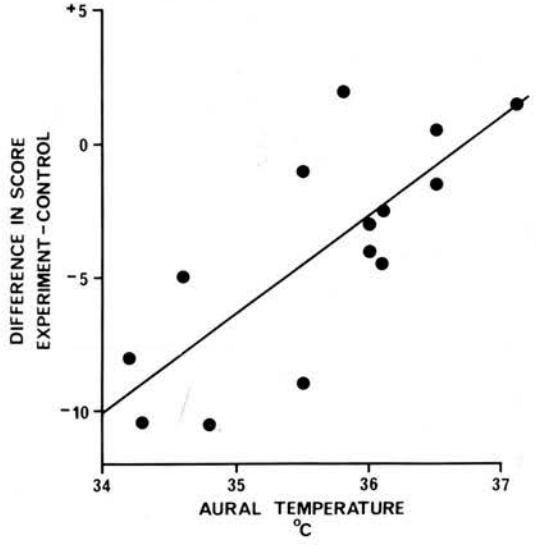

FIG. 2. Facts subsequently recalled at normal body temperatures in relation to body temperatures present when they were memorized after end of cold immersions. Number recalled is given as difference from number recalled by same subject in prior control test in which both memorization and recall took place at normal body temperature (mean no. of facts recalled in controls, 12.5). Decrement of facts registered = 3.7 (36.7 − body temperature, °C).

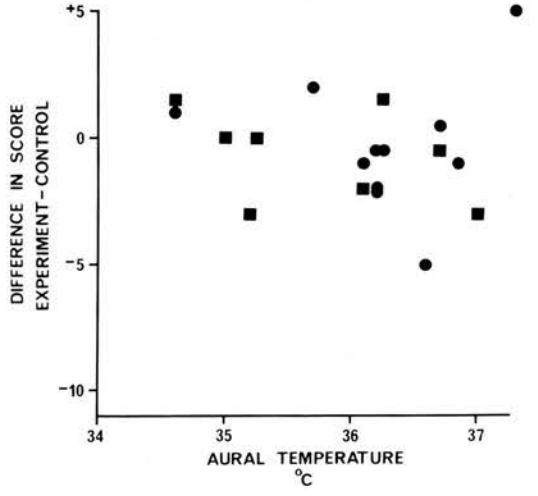

FIG. 3. Facts recalled in relation to body temperatures at time of recall after end of cold immersions with facts previously memorized at normal body temperature. Number recalled is given as difference from number recalled by same subject in control tests, given either before (●) or after (■) immersion, in which both memorization and recall took place at normal body temperature (mean no. of facts recalled in controls, 12.0).

Calculation and reasoning tests at reduced deep body temperature. Figures 4 and 5 show the time taken by 17 subjects to perform a double-digit calculation test and a reasoning test at reduced deep body temperature. The tests were performed shortly after the subject left the water at 15°C and entered the warm bath at 41°C, when sensations of cold and shivering had ceased. The time taken to complete each test is expressed as a percentage of the time needed by the subject to perform a similar test in the warm water after restoration of normal body temperature (±0.3°C). There was a tendency for the subjects with lower body temperatures to take longer in completing the tests. This was most clearly seen in the relationship between body temperature and performance of the double-digit calculation test ($r = -0.72$, $P < 0.01$). The tendency to slowing in the reasoning test did not reach statistical significance ($r = -0.40$, $P < 0.2$) because the degree of slowing at reduced body temperature was quite variable. One subject at a body temperature of 34.6°C took 434% of his control time to complete the test, an order of magnitude greater than was seen with other subjects who had cooled to similar temperatures. No relationship was found between time for performance of the single-digit calculation test and deep body temperature ($r = -0.25$), and mean performance time for this test was very close to that taken after restoration of normal body temperature. The time taken to complete the tests at reduced body temperature in the warm bath was 122.2 ± 18.6 s in the double-digit calculation (at 35.7 ± 0.2°C), 55.4 ± 12.6 s in the reasoning test (at 35.7 ± 0.2°C), and 67.3 ± 9.4 s in the single-digit calculation (at 35.7 ± 0.02°C). In the tests made after restoration of normal body temperature, which were used as a reference for tests at reduced body temperature, the time taken to complete the tests was 96.7 ± 13.5 s in the double-digit calculation at 37.4 ± 0.1°C, 34.5 ± 4.9 s in the reasoning test at 37.4 ± 0.1°C, and 63.2 ± 6.5 s in the single-digit calculation at 37.2 ± 0.1°C. There was no significant relationship between body temperature and time to complete any of the three tests at that time ($r < 0.45$). The variability of individual times in all of these tests made it necessary to analyze changes as a percentage of the individual's reference time.

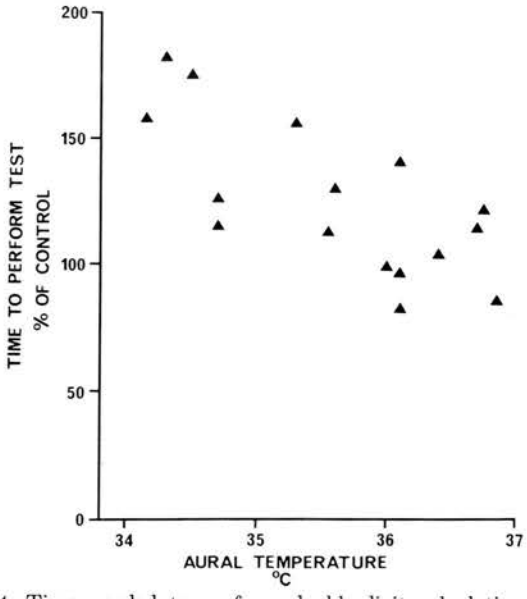

FIG. 4. Time needed to perform double-digit calculation test in relation to core temperature. Time is % of time taken by same subject in a subsequent control test (mean for controls, 98.0 s) at normal body temperature.

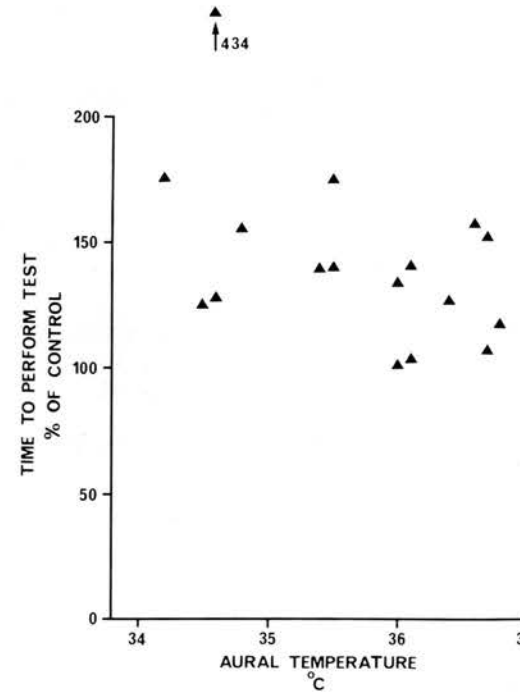

FIG. 5. Time needed to perform A-B reasoning test in relation to core temperature. Time is % of time taken by same subject in subsequent control test (mean for controls, 34.5 s) at normal body temperature.

A separate series of control tests of calculation and reasoning was carried out on another group of six subjects at equivalent time intervals, but under control conditions in air at normal body temperature throughout. The times needed to perform the tests equivalent to the test performed at reduced body temperature in the warm bath were 87.7 ± 10.9 s for the double-digit calculation, 21.4 ± 2.2 s for the reasoning test, and 42.2 ± 1.8 s for the single-digit calculation. None of these values was significantly different from the time needed to perform the final test, equivalent to the reference test in the warm bath at normal body temperature: 89.5 ± 11.0 s for the double-digit calculation, 20.8 ± 2.1 s for the reasoning test, and 57.4 ± 3.0 s for the single-digit calculation. This indicates that training effects had been virtually eliminated by the fifth and sixth tests which were used for comparison.

The number of correct answers obtained in the tests, unlike the time needed to perform them, showed no clear relation to body temperature. In the tests made just after the subjects left the cold for the warm bath, the correlation between body temperature and number of correct answers (as difference from number of correct answers obtained after body temperature rose to normal) was $r = -0.13$ for the double-digit calculation, $r = 0.50$ for the reasoning test, and $r = -0.14$ for the single-digit calculation test. Nor did the number of correct answers then

differ significantly from the scores after body rewarming. In the warm bath, before and after rewarming of body core, the numbers of correct answers were respectively 4.7 ± 0.3 and 4.7 ± 0.3, (maximum possible score = 6) for the double-digit calculation, 7.0 ± 0.3 and 6.5 ± 0.3 (maximum possible score = 8) for the reasoning test, and 9.5 ± 0.2 and 9.5 ± 0.2 (maximum possible score = 10) for the single-digit calculation ($n = 6$). Nor did numbers of correct scores differ significantly between the fifth and sixth tests on six control subjects at normal body temperature throughout. They were respectively 4.5 ± 0.6 and 4.3 ± 0.7, for double-digit calculations, 7.8 ± 0.2 and 7.8 ± 0.2 for reasoning tests, and 9.7 ± 0.2 and 9.8 ± 0.2 for single-digit calculations. ($n = 6$).

DISCUSSION

The most striking finding was the impairment of memory registration which was observed as body temperatures fell from about 36.7°C and that was severe at body temperatures around 35°C. Body core temperatures well below 36°C, and sometimes below 35°C, have been recorded in healthy young people at the end of recreational cold swims (15) and in commercial divers using conventional warm-water-heated suits in the North Sea (12). Temperatures as low as 35.6°C have occasionally been reported in the absence of cold exposure, in the early morning, at the low point of the diurnal temperature variation (17).

The fact that the impairment of registration was found even when skin temperature was raised sufficiently to eliminate cold discomfort makes it likely that the impairment was caused directly by the low temperature of the brain. The aural canal temperature with servo-controlled heating of the outer ear, used to measure core temperature, can be taken as an accurate indication of the temperature of the blood perfusing the brain, since it closely follows esophageal temperature (13). In contrast, memory recall seemed to be unimpaired by lowering core temperature to even 34.6°C. One obvious practical implication of our results is that divers cannot be expected to give a reliable account afterward of events that took place during a dive in which their body temperature was low. Anecdotal accounts of North Sea diving suggest that, in practice, reports of underwater inspections given after completion of the dives have often proved unreliable. Our results also suggest that mild hypothermia could be a serious hazard to a diver who, for example, must memorize during a dive a route that will bring him out safely from underwater structures.

Although lowered body core temperature also slowed the performance of a complex calculation task, and probably slowed performance of a reasoning task, the slowing was not accompanied by any loss of accuracy provided that adequate time was allowed for the task to be completed. The slowing, however, was substantial, amounting to about 175% for the calculation at a core temperature of 34.2°C. One factor in this is likely to be slowing of synaptic transmission; a fall in temperature of 15°C from 37°C has been shown to increase the rise time of the end-plate potential in cat muscle to 200% and to increase the fall time to 260% of control values (4). Such delays are probably compounded by other factors in complex mental tasks, such as the two-digit calculation task, since we observed almost as much slowing with a fall in body core temperature of only approximately 3°C. From the practical viewpoint, the slowing itself could be a hazard when quick responses are needed in emergencies such as diving often presents.

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REFERENCES

1. BADDELEY, A. D. A three-minute reasoning test based on grammatical transformation. *Psychonomic Sci.* 10: 341–342, 1968.
2. BADDELEY, A. D., W. J. CUCCARO, G. H. EGSTROM, G. WELTMAN, AND M. A. WILLIS. Cognitive efficiency of divers working in cold water. *Hum. Factors* 17: 446–454, 1975.
3. BOWEN, H. M. Diver performance and the effects of cold. *Hum. Factors* 10: 445–464, 1968.
4. BOYD, I. A., AND A. R. MARTIN. The end-plate potential in mammalian muscle. *J. Physiol. London* 132: 74–91, 1956.
5. COLESHAW, S. R. K., W. R. KEATINGE, P. J. MINCER, AND R. N. M. VAN SOMEREN. Thermoregulatory response to cooling of hands and feet (Abstract). *J. Physiol. London* 325: 38P, 1982.
6. CURRIE, J. *Medical Reports on the Effects of Water Cold and Warm.* London: Cadell & Davies, 1798.
7. DAVIS, F. M., A. D. BADDELEY, AND T. R. HANCOCK. Diver performance: the effect of cold. *Undersea Biomed. Res.* 2: 195–213, 1975.
8. FRIEDMAN, M. P., AND F. L. GREITZER. Organization and study time in learning from reading. *J. Educ. Psychol.* 63: 609–616, 1972.
9. HAYWARD, M. G., AND W. R. KEATINGE. Progressive symptomless hypothermia in water: possible cause of diving accidents. *Br. Med. J.* 1: 1182, 1979.
10. KEATINGE, W. R. *The Effect of Work, Clothing and Adaptation on the Maintenance of the Body Temperature in Water And on Reflex Responses to Immersion.* (PhD thesis, Cambridge, UK. Univ. of Cambridge, 1959.
11. KEATINGE, W. R. The effects of subcutaneous fat and of previous exposure to cold on the body temperature, peripheral blood flow and metabolic rate of men in cold water. *J. Physiol. London* 153: 166–178, 1960.
12. KEATINGE, W. R., M. G. HAYWARD, AND N. K. I. MCIVER. Hypothermia during saturation diving in the North Sea. *Br. Med. J.* 1: 291, 1980.
13. KEATINGE, W. R., AND R. E. G. SLOAN. Deep body temperature from aural canal with servo-controlled heating to outer ear. *J. Appl. Physiol.* 38: 919–921, 1975.
14. LAUFMAN, H. Profound accidental hypothermia. *J. Am. Med. Assoc.* 147: 1201–1212, 1951.
15. SLOAN, R. E. G., AND W. R. KEATINGE. Cooling rates of young people swimming in cold water. *J. Appl. Physiol.* 35: 371–375, 1973.
16. STANG, P. R., AND E. L. WEINER. Diver performance in cold water. *Hum. Factors* 12: 391–399, 1970.
17. VAN LOON, J. H. Diurnal body temperature curves in shift workers. *Ergonomics* 6: 267–273, 1963.
18. VAN SOMEREN, R. N. M., S. R. K. COLESHAW, P. J. MINCER, AND W. R. KEATINGE. Restoration of thermoregulatory response to body cooling by cooling hands and feet. *J. Appl. Physiol.: Respirat. Environ. Exercise* 53: 1228–1233, 1982.

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Memory registration at mildly depressed deep body temperatures

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Impairment of mental performance has often been observed in divers working in cold water (e.g. Bowen, 1968; Stang & Weiner, 1970; Baddeley, Cuccaro, Egstrom, Weltman & Willis, 1975; Davis, Baddeley & Hancock, 1975). However, the impairment was often attributed to the distracting effect of cold skin. We recently reported that a North Sea diver using the hot-water flooding system for heating (Keatinge, Hayward & McIver, 1980) and a volunteer immersed in lukewarm water (Hayward & Keatinge, 1979) suffered falls in deep body temperature to below 35 °C without any major sensation of cold. We have now measured the effect of similar falls in deep body temperature, but with warm skin, on mental performance of volunteers.

Deep body temperatures were reduced by immersion in water at 15 °C. The subjects then went into a warm bath (41 °C), and as soon as they felt comfortable and the skin warm, were asked to memorise a test passage, containing fifteen facts, in $2\frac{1}{2}$ min, which they tried to recall later on rewarming to normal body temperature. A significant relationship was found between decrement in memory registration and deep body temperature; at body temperatures below 35 °C, recall ranged from 17–43 % of performance in a similar test at normal body temperature throughout. By contrast, there was no reduction in ability to recall, at low deep body temperature, facts which had been learned at normal body temperature immediately before immersion. Nor was there any decrease in the accuracy of performance of brief calculation and reasoning tasks at the low body temperature. The speed of performance of the more complicated of these tasks (e.g. double-digit addition) was, however, reduced at body temperatures below 35 °C.

These deficiencies in memory registration and in speed of reasoning at reduced deep body temperatures seem likely to have been contributory factors to diving accidents.

REFERENCES

Baddeley, A. D., Cuccaro, W. J., Egstrom, G. H., Weltman, G. & Willis, M. A. (1975). *Human Factors* **17**, 446–454.
Bowen, H. M. (1968). *Human Factors* **10**, 445–464.
Davis, F. M., Baddeley, A. D. & Hancock, T. R. (1975). *Undersea Biomed. Res.* **2**, 195–213.
Hayward, M. G. & Keatinge, W. R. (1979). *Brit. med. J.* i, 1182.
Keatinge, W. R., Hayward, M. G. & McIver, N. K. I. (1980). *Brit. med. J.* i, 291.
Stang, P. R. & Weiner, E. L. (1970). *Human Factors* **12**, 391–399.